```
=> file hcaplus; d que l24; d que l27; d que l29; d que l30
FILE 'HCAPLUS' ENTERED AT 13:22:43 ON 31 AUG 2004
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FILE COVERS 1907 - 31 Aug 2004  VOL 141 ISS 10
FILE LAST UPDATED: 30 Aug 2004  (20040830/ED)

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.
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L2	37296	SEA FILE=CAPLUS ABB=ON PLU=ON HORSE OR PONY OR EQUUS
L3	107364	SEA FILE=CAPLUS ABB=ON PLU=ON COW OR CATTLE OR BOS
L4	79712	SEA FILE=CAPLUS ABB=ON PLU=ON SHEEP OR LAMB OR OVIS
L5	68661	SEA FILE=CAPLUS ABB=ON PLU=ON CAT OR FELIS OR FELIDAE
L6	142758	SEA FILE=CAPLUS ABB=ON PLU=ON DOG OR CANIS
L7	231361	SEA FILE=CAPLUS ABB=ON PLU=ON PIG OR SWINE OR SUS
L8	20451	SEA FILE=CAPLUS ABB=ON PLU=ON GOAT OR CAPRA
L10	10140	SEA FILE=HCAPLUS ABB=ON PLU=ON AUTOIMMUNE DISEASE+PFT/CT
L11	910	SEA FILE=HCAPLUS ABB=ON PLU=ON LYMPHOPROLIFERATIVE DISORDERS+ PFT/CT
L12	33348	SEA FILE=HCAPLUS ABB=ON PLU=ON (MAST OR MYELOID OR PLASMA) (W) (CELL) OR (B CELL OR T CELL) (2A) (DISEASE OR DISORDER)
L13	678572	SEA FILE=HCAPLUS ABB=ON PLU=ON ?LYMPHOMA? OR ?LEUKEM? OR ?MYELOMA? OR ?MASTOCYTOM? OR ?CANCER? OR ?MALIGN? OR ?CARCINOM? OR ?TUMOR? OR ?TUMOUR?
L14	180612	SEA FILE=HCAPLUS ABB=ON PLU=ON ANTITUMOR AGENTS+OLD/CT
L15	10124	SEA FILE=HCAPLUS ABB=ON PLU=ON IMMUNOTHERAPY+PFT/CT
L16	18256	SEA FILE=HCAPLUS ABB=ON PLU=ON IMMUNE THERAPY OR IMMUNOMODULA T?
L17	48408	SEA FILE=HCAPLUS ABB=ON PLU=ON "ANTIBODIES AND IMMUNOGLOBULIN S"/CT
L18	16783	SEA FILE=HCAPLUS ABB=ON PLU=ON "FUSION PROTEINS (CHIMERIC PROTEINS)"+PFT/CT
L19	147298	SEA FILE=HCAPLUS ABB=ON PLU=ON CYTOKINE OR IMMUNOCONJUGATE
L21	231	SEA FILE=HCAPLUS ABB=ON PLU=ON (L2 OR L3 OR L4 OR L5 OR L6 OR L7 OR L8) AND (L10 OR L11 OR L12 OR L13) AND (L14 OR L15 OR L16) AND L17
L22	91	SEA FILE=HCAPLUS ABB=ON PLU=ON (L2 OR L3 OR L4 OR L5 OR L6 OR L7 OR L8) AND (L10 OR L11 OR L12 OR L13) AND (L14 OR L15 OR L16) AND L18
L23	294	SEA FILE=HCAPLUS ABB=ON PLU=ON (L2 OR L3 OR L4 OR L5 OR L6 OR L7 OR L8) AND (L10 OR L11 OR L12 OR L13) AND (L14 OR L15 OR L16) AND L19
L24	1	SEA FILE=HCAPLUS ABB=ON PLU=ON NAKED AND (L21 OR L22 OR L23)

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L2        37296 SEA FILE=CAPLUS ABB=ON  PLU=ON  HORSE OR PONY OR EQUUS
L3       107364 SEA FILE=CAPLUS ABB=ON  PLU=ON  COW OR CATTLE OR BOS
L4        79712 SEA FILE=CAPLUS ABB=ON  PLU=ON  SHEEP OR LAMB OR OVIS
L5        68661 SEA FILE=CAPLUS ABB=ON  PLU=ON  CAT OR FELIS OR FELIDAE
L6       142758 SEA FILE=CAPLUS ABB=ON  PLU=ON  DOG OR CANIS
L7       231361 SEA FILE=CAPLUS ABB=ON  PLU=ON  PIG OR SWINE OR SUS
L8        20451 SEA FILE=CAPLUS ABB=ON  PLU=ON  GOAT OR CAPRA
L10       10140 SEA FILE=HCAPLUS ABB=ON  PLU=ON  AUTOIMMUNE DISEASE+PFT/CT
L11         910 SEA FILE=HCAPLUS ABB=ON  PLU=ON  LYMPHOPROLIFERATIVE DISORDERS+
                PFT/CT
L12       33348 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (MAST OR MYELOID OR PLASMA)
                (W) (CELL) OR (B CELL OR T CELL) (2A) (DISEASE OR DISORDER)
L13      678572 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ?LYMPHOMA? OR ?LEUKEM? OR
                ?MYELOMA? OR ?MASTOCYTOM? OR ?CANCER? OR ?MALIGN? OR ?CARCINOM?
                 OR ?TUMOR? OR ?TUMOUR?
L15       10124 SEA FILE=HCAPLUS ABB=ON  PLU=ON  IMMUNOTHERAPY+PFT/CT
L17       48408 SEA FILE=HCAPLUS ABB=ON  PLU=ON  "ANTIBODIES AND IMMUNOGLOBULIN
                S"/CT
L25          61 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (L2 OR L3 OR L4 OR L5 OR L6
                OR L7 OR L8) AND (L10 OR L11 OR L12 OR L13) AND L15 AND L17
L27           5 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L25 AND PRY<2000


L2        37296 SEA FILE=CAPLUS ABB=ON  PLU=ON  HORSE OR PONY OR EQUUS
L3       107364 SEA FILE=CAPLUS ABB=ON  PLU=ON  COW OR CATTLE OR BOS
L4        79712 SEA FILE=CAPLUS ABB=ON  PLU=ON  SHEEP OR LAMB OR OVIS
L5        68661 SEA FILE=CAPLUS ABB=ON  PLU=ON  CAT OR FELIS OR FELIDAE
L6       142758 SEA FILE=CAPLUS ABB=ON  PLU=ON  DOG OR CANIS
L7       231361 SEA FILE=CAPLUS ABB=ON  PLU=ON  PIG OR SWINE OR SUS
L8        20451 SEA FILE=CAPLUS ABB=ON  PLU=ON  GOAT OR CAPRA
L10       10140 SEA FILE=HCAPLUS ABB=ON  PLU=ON  AUTOIMMUNE DISEASE+PFT/CT
L11         910 SEA FILE=HCAPLUS ABB=ON  PLU=ON  LYMPHOPROLIFERATIVE DISORDERS+
                PFT/CT
L12       33348 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (MAST OR MYELOID OR PLASMA)
                (W) (CELL) OR (B CELL OR T CELL) (2A) (DISEASE OR DISORDER)
L13      678572 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ?LYMPHOMA? OR ?LEUKEM? OR
                ?MYELOMA? OR ?MASTOCYTOM? OR ?CANCER? OR ?MALIGN? OR ?CARCINOM?
                 OR ?TUMOR? OR ?TUMOUR?
L14      180612 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ANTITUMOR AGENTS+OLD/CT
L17       48408 SEA FILE=HCAPLUS ABB=ON  PLU=ON  "ANTIBODIES AND IMMUNOGLOBULIN
                S"/CT
L18       16783 SEA FILE=HCAPLUS ABB=ON  PLU=ON  "FUSION PROTEINS (CHIMERIC
                PROTEINS)"+PFT/CT
L28         237 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (L2 OR L3 OR L4 OR L5 OR L6
                OR L7 OR L8) AND (L10 OR L11 OR L12 OR L13) AND L14 AND (L17
                OR L18)
L29          14 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L28 AND PY<2000


L2        37296 SEA FILE=CAPLUS ABB=ON  PLU=ON  HORSE OR PONY OR EQUUS
L3       107364 SEA FILE=CAPLUS ABB=ON  PLU=ON  COW OR CATTLE OR BOS
L4        79712 SEA FILE=CAPLUS ABB=ON  PLU=ON  SHEEP OR LAMB OR OVIS
L5        68661 SEA FILE=CAPLUS ABB=ON  PLU=ON  CAT OR FELIS OR FELIDAE
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L6       142758 SEA FILE=CAPLUS ABB=ON  PLU=ON  DOG OR CANIS
L7       231361 SEA FILE=CAPLUS ABB=ON  PLU=ON  PIG OR SWINE OR SUS
L8        20451 SEA FILE=CAPLUS ABB=ON  PLU=ON  GOAT OR CAPRA
L10       10140 SEA FILE=HCAPLUS ABB=ON  PLU=ON  AUTOIMMUNE DISEASE+PFT/CT
L11         910 SEA FILE=HCAPLUS ABB=ON  PLU=ON  LYMPHOPROLIFERATIVE DISORDERS+
                PFT/CT
L12       33348 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (MAST OR MYELOID OR PLASMA)
                (W) (CELL) OR (B CELL OR T CELL) (2A) (DISEASE OR DISORDER)
L13      678572 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ?LYMPHOMA? OR ?LEUKEM? OR
                ?MYELOMA? OR ?MASTOCYTOM? OR ?CANCER? OR ?MALIGN? OR ?CARCINOM?
                 OR ?TUMOR? OR ?TUMOUR?
L14      180612 SEA FILE=HCAPLUS ABB=ON  PLU=ON  ANTITUMOR AGENTS+OLD/CT
L17       48408 SEA FILE=HCAPLUS ABB=ON  PLU=ON  "ANTIBODIES AND IMMUNOGLOBULIN
                S"/CT
L18       16783 SEA FILE=HCAPLUS ABB=ON  PLU=ON  "FUSION PROTEINS (CHIMERIC
                PROTEINS)"+PFT/CT
L28         237 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (L2 OR L3 OR L4 OR L5 OR L6
                OR L7 OR L8) AND (L10 OR L11 OR L12 OR L13) AND L14 AND (L17
                OR L18)
L30          39 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L28 AND PRY<2000


=> s l24 or l27 or l29 or l30
L108         42 L24 OR L27 OR L29 OR L30

=> file medline; d que 146
FILE 'MEDLINE' ENTERED AT 13:23:10 ON 31 AUG 2004

 FILE LAST UPDATED: 28 AUG 2004 (20040828/UP).  FILE COVERS 1951 TO DATE.

 On February 29, 2004, the 2004 MeSH terms were loaded.  See HELP RLOAD
 for details. OLDMEDLINE now back to 1951.

 MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the
 MeSH 2004 vocabulary.  See http://www.nlm.nih.gov/mesh/ and
 http://www.nlm.nih.gov/pubs/techbull/nd03/nd03_mesh.html for a
 description of changes.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.


L2        37296 SEA FILE=CAPLUS ABB=ON  PLU=ON  HORSE OR PONY OR EQUUS
L3       107364 SEA FILE=CAPLUS ABB=ON  PLU=ON  COW OR CATTLE OR BOS
L4        79712 SEA FILE=CAPLUS ABB=ON  PLU=ON  SHEEP OR LAMB OR OVIS
L5        68661 SEA FILE=CAPLUS ABB=ON  PLU=ON  CAT OR FELIS OR FELIDAE
L6       142758 SEA FILE=CAPLUS ABB=ON  PLU=ON  DOG OR CANIS
L7       231361 SEA FILE=CAPLUS ABB=ON  PLU=ON  PIG OR SWINE OR SUS
L8        20451 SEA FILE=CAPLUS ABB=ON  PLU=ON  GOAT OR CAPRA
L33      948603 SEA FILE=MEDLINE ABB=ON  PLU=ON  (L2 OR L3 OR L4 OR L5 OR L6
                OR L7 OR L8)
L34      240806 SEA FILE=MEDLINE ABB=ON  PLU=ON  AUTOIMMUNE DISEASES+NT/CT
L35      257835 SEA FILE=MEDLINE ABB=ON  PLU=ON  LYMPHOPROLIFERATIVE DISORDERS+
                NT/CT
L36     1534935 SEA FILE=MEDLINE ABB=ON  PLU=ON  NEOPLASMS+NT/CT
L39      603117 SEA FILE=MEDLINE ABB=ON  PLU=ON  ANTINEOPLASTIC AGENTS+NT/CT
L40      154307 SEA FILE=MEDLINE ABB=ON  PLU=ON  IMMUNOSUPPRESSIVE AGENTS+NT/CT

L41       42096 SEA FILE=MEDLINE ABB=ON  PLU=ON  RECOMBINANT FUSION PROTEINS/CT
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L43       511992 SEA FILE=MEDLINE ABB=ON  PLU=ON  ANTIBODIES+NT/CT
L46            4 SEA FILE=MEDLINE ABB=ON  PLU=ON  L33 AND (L34 OR L35 OR L36)
                 AND L43 AND (L39 OR L40) AND L41


=> file biosis; d que l71
FILE 'BIOSIS' ENTERED AT 13:23:19 ON 31 AUG 2004
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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 26 August 2004 (20040826/ED)

FILE RELOADED: 19 October 2003.


L2         37296 SEA FILE=CAPLUS ABB=ON  PLU=ON  HORSE OR PONY OR EQUUS
L3        107364 SEA FILE=CAPLUS ABB=ON  PLU=ON  COW OR CATTLE OR BOS
L4         79712 SEA FILE=CAPLUS ABB=ON  PLU=ON  SHEEP OR LAMB OR OVIS
L5         68661 SEA FILE=CAPLUS ABB=ON  PLU=ON  CAT OR FELIS OR FELIDAE
L6        142758 SEA FILE=CAPLUS ABB=ON  PLU=ON  DOG OR CANIS
L7        231361 SEA FILE=CAPLUS ABB=ON  PLU=ON  PIG OR SWINE OR SUS
L8         20451 SEA FILE=CAPLUS ABB=ON  PLU=ON  GOAT OR CAPRA
L57       911632 SEA FILE=BIOSIS ABB=ON  PLU=ON  (L2 OR L3 OR L4 OR L5 OR L6 OR
                 L7 OR L8)
L58        23190 SEA FILE=BIOSIS ABB=ON  PLU=ON  AUTOIMMUNE (W) (DISEASE OR
                 DISORDER)
L59         6318 SEA FILE=BIOSIS ABB=ON  PLU=ON  LYMPHOPROLIFERATIVE (W)
                 (DISEASE OR DISORDER)
L60        52226 SEA FILE=BIOSIS ABB=ON  PLU=ON  (MAST OR MYELOID OR PLASMA)
                 (W) (CELL) OR (B CELL OR T CELL) (2A) (DISEASE OR DISORDER)
L61      1480198 SEA FILE=BIOSIS ABB=ON  PLU=ON  ?CANCER? OR ?MALIGN? OR
                 ?CARCINOM? OR ?TUMOR? OR ?TUMOUR? OR ?NEOPLA? OR ?CARCINOM?
L62       282078 SEA FILE=BIOSIS ABB=ON  PLU=ON  ANTITUMOR? OR IMMUNOTHERAPY OR
                 IMMUNE THERAPY OR ANTINEOPLAST?
L63       559493 SEA FILE=BIOSIS ABB=ON  PLU=ON  ANTIBOD?
L66          460 SEA FILE=BIOSIS ABB=ON  PLU=ON  L57 AND (L58 OR L59 OR L60 OR
                 L61) AND L62 AND L63
L68       401791 SEA FILE=BIOSIS ABB=ON  PLU=ON  DOG OR CAT OR HORSE OR CANIS
                 OR FELIDAE OR FELIS OR EQUUS
L69          115 SEA FILE=BIOSIS ABB=ON  PLU=ON  L66 AND L68
L70           90 SEA FILE=BIOSIS ABB=ON  PLU=ON  L69 AND PY<2000
L71           24 SEA FILE=BIOSIS ABB=ON  PLU=ON  L70 AND (CANINE OR FELINE OR
                 THYMUS OR EQUINE OR MYELOABLAT? OR MONONCLEAR OR ANTIGANGLIO?
                 OR  BEAGLE OR LACTAMASE)/TI


=> file embase; d que 191; d que 194
FILE 'EMBASE' ENTERED AT 13:23:31 ON 31 AUG 2004
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 FILE COVERS 1974 TO 26 Aug 2004 (20040826/ED)

 EMBASE has been reloaded. Enter HELP RLOAD for details.

 This file contains CAS Registry Numbers for easy and accurate
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substance identification.

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L72      251301 SEA FILE=EMBASE ABB=ON  PLU=ON  DOG OR CAT OR HORSE OR CANINE
                OR FELINE OR EQUUS OR CANIS OR FELIS OR FELIDAE
L73      158261 SEA FILE=EMBASE ABB=ON  PLU=ON  AUTOIMMUNE DISEASE+ALL/CT
L74      129386 SEA FILE=EMBASE ABB=ON  PLU=ON  LYMPHOPROLIFERATIVE DISEASE+ALL
                /CT
L75     1244309 SEA FILE=EMBASE ABB=ON  PLU=ON  CANCER? OR MALIGN? OR CARCINOM?
                 OR TUMOR? OR TUMOUR? OR NEOPLAS?
L77       54338 SEA FILE=EMBASE ABB=ON  PLU=ON  IMMUNOTHERAPY+NT/CT
L78      103044 SEA FILE=EMBASE ABB=ON  PLU=ON  ANTITUMOR OR ANTINEOPLAS?
L80      612057 SEA FILE=EMBASE ABB=ON  PLU=ON  ANTIBOD? OR IMMUNOCONJUG? OR
                CYTOKINE
L82         151 SEA FILE=EMBASE ABB=ON  PLU=ON  L72 AND (L73 OR L74 OR L75)
                AND L80 AND (L77 OR L78)
L84         101 SEA FILE=EMBASE ABB=ON  PLU=ON  L82 AND PY<2000
L85      196365 SEA FILE=EMBASE ABB=ON  PLU=ON  DOG/CT OR CAT/CT OR HORSE/CT
L89       24763 SEA FILE=EMBASE ABB=ON  PLU=ON  L85/MAJ
L90           5 SEA FILE=EMBASE ABB=ON  PLU=ON  L84 AND L89
L91           2 SEA FILE=EMBASE ABB=ON  PLU=ON  L90 AND (EQUINE OR IMMUNOCHEM?)
                /TI



L72      251301 SEA FILE=EMBASE ABB=ON  PLU=ON  DOG OR CAT OR HORSE OR CANINE
                OR FELINE OR EQUUS OR CANIS OR FELIS OR FELIDAE
L73      158261 SEA FILE=EMBASE ABB=ON  PLU=ON  AUTOIMMUNE DISEASE+ALL/CT
L74      129386 SEA FILE=EMBASE ABB=ON  PLU=ON  LYMPHOPROLIFERATIVE DISEASE+ALL
                /CT
L75     1244309 SEA FILE=EMBASE ABB=ON  PLU=ON  CANCER? OR MALIGN? OR CARCINOM?
                 OR TUMOR? OR TUMOUR? OR NEOPLAS?
L77       54338 SEA FILE=EMBASE ABB=ON  PLU=ON  IMMUNOTHERAPY+NT/CT
L78      103044 SEA FILE=EMBASE ABB=ON  PLU=ON  ANTITUMOR OR ANTINEOPLAS?
L80      612057 SEA FILE=EMBASE ABB=ON  PLU=ON  ANTIBOD? OR IMMUNOCONJUG? OR
                CYTOKINE
L82         151 SEA FILE=EMBASE ABB=ON  PLU=ON  L72 AND (L73 OR L74 OR L75)
                AND L80 AND (L77 OR L78)
L84         101 SEA FILE=EMBASE ABB=ON  PLU=ON  L82 AND PY<2000
L92          35 SEA FILE=EMBASE ABB=ON  PLU=ON  L84 AND ?ANTIGEN?
L94           9 SEA FILE=EMBASE ABB=ON  PLU=ON  L92 AND (RADIOLABEL? OR
                SUPERANT? OR T268G OR ALLOGEN? OR MYASTHENIA OR EXTRACORP? OR
                LYSIS)/TI


=> s l91 or l94
L109         11 L91 OR L94

=> file wpix; d que l107
FILE 'WPIX' ENTERED AT 13:23:54 ON 31 AUG 2004
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FILE LAST UPDATED:          26 AUG 2004      <20040826/UP>
MOST RECENT DERWENT UPDATE:     200455        <200455/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

>>> FOR A COPY OF THE DERWENT WORLD PATENTS INDEX STN USER GUIDE,
    PLEASE VISIT:
 http://www.stn-international.de/training_center/patents/stn_guide.pdf <<<
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http://thomsonderwent.com/coverage/latestupdates/ <<<

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GUIDES, PLEASE VISIT:
http://thomsonderwent.com/support/userguides/ <<<

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DOCUMENTATION NOW AVAILABLE IN DERWENT WORLD PATENTS INDEX
FIRST VIEW - FILE WPIFV.
FOR FURTHER DETAILS: http://www.thomsonderwent.com/dwpifv <<<

>>> NEW DISPLAY FORMAT HITSTR ADDED ALLOWING DISPLAY OF
HIT STRUCTURES WITHIN THE BIBLIOGRAPHIC DOCUMENT <<<

L95 35960 SEA FILE=WPIX ABB=ON PLU=ON CAT OR DOG OR HORSE OR CANINE OR FELINE OR EQUUS OR FELIDAE OR CANIS OR PONY
L96 63881 SEA FILE=WPIX ABB=ON PLU=ON ANTIBOD?
L98 63937 SEA FILE=WPIX ABB=ON PLU=ON (AUTOIMMUNE OR LYMPHOPROLIFERATIVE) (W) DISEASE OR DISORDER
L99 2198 SEA FILE=WPIX ABB=ON PLU=ON (MAST OR MYELOID OR PLASMA) (W) (CELL) OR (B CELL OR T CELL) (2A) (DISEASE OR DISORDER)
L100 91814 SEA FILE=WPIX ABB=ON PLU=ON CANCER? OR NEOPL? OR CARCINO? OR TUMOR? OR TUMOUR?
L101 18508 SEA FILE=WPIX ABB=ON PLU=ON ANTINEOPL? OR ANTITUMOR? OR ANTICARCINO? OR ANTITUMOUR OR ANTI (W) (CANCER? OR NEOPL? OR CARCINO? OR TUMOR? OR TUMOUR?)
L102 1573 SEA FILE=WPIX ABB=ON PLU=ON IMMUNE THERAPY OR IMMUNOTHERAPY
L104 59 SEA FILE=WPIX ABB=ON PLU=ON L95 AND L96 AND (L98 OR L99 OR L100) AND L101
L105 13 SEA FILE=WPIX ABB=ON PLU=ON L95 AND L96 AND (L98 OR L99 OR L100) AND L102
L106 33 SEA FILE=WPIX ABB=ON PLU=ON (L104 OR L105) AND PRY<2000
L107 5 SEA FILE=WPIX ABB=ON PLU=ON L106 AND (GENIGN OR POXV? OR CATS OR ANIMAL? OR ANTIGENS)/TI

=> dup rem l46 l108 l71 l109 l107
FILE 'MEDLINE' ENTERED AT 13:24:18 ON 31 AUG 2004

FILE 'HCAPLUS' ENTERED AT 13:24:18 ON 31 AUG 2004
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FILE 'WPIX' ENTERED AT 13:24:18 ON 31 AUG 2004
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PROCESSING COMPLETED FOR L46
PROCESSING COMPLETED FOR L108
PROCESSING COMPLETED FOR L71
PROCESSING COMPLETED FOR L109

PROCESSING COMPLETED FOR L107
L110 84 DUP REM L46 L108 L71 L109 L107 (2 DUPLICATES REMOVED)
ANSWERS '1-4' FROM FILE MEDLINE
ANSWERS '5-46' FROM FILE HCAPLUS
ANSWERS '47-70' FROM FILE BIOSIS
ANSWERS '71-79' FROM FILE EMBASE
ANSWERS '80-84' FROM FILE WPIX

=> d ibib ed ab l110 1-79; d ibib ab abex l110 80-84

L110 ANSWER 1 OF 84 MEDLINE on STN
ACCESSION NUMBER: 2001364658 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11418697
TITLE: A retro-inverso peptide mimic of CD28 encompassing the MYPPPY motif adopts a polyproline type II helix and inhibits encephalitogenic T cells in vitro.
AUTHOR: Srinivasan M; Wardrop R M; Gienapp I E; Stuckman S S; Whitacre C C; Kaumaya P T
CORPORATE SOURCE: Department of Microbiology, College of Biological Sciences, Ohio State University, Columbus, OH 43210, USA.
CONTRACT NUMBER: RO1 AI40302 (NIAID)
RO1 AI43376 (NIAID)
SOURCE: Journal of immunology (Baltimore, Md. : 1950), (2001 Jul 1) 167 (1) 578-85.
Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200109
ENTRY DATE: Entered STN: 20010924
Last Updated on STN: 20010924
Entered Medline: 20010920
ED Entered STN: 20010924
Last Updated on STN: 20010924
Entered Medline: 20010920
AB Complete activation of T cells requires two signals: an Ag-specific signal delivered via the TCR by the peptide-MHC complex and a second costimulatory signal largely provided by B7:CD28/CTLA-4 interactions. Previous studies have shown that B7 blockade can either ameliorate experimental autoimmune encephalomyelitis by interfering with CD28 signaling or exacerbate the disease by concomitant blockade of CTLA-4 interaction. Therefore, we developed a functional CD28 mimic to selectively block B7:CD28 interactions. The design, synthesis, and structural and functional properties of the CD28 free peptide, the end group-blocked CD28 peptide, and its retro-inverso isomer are shown. The synthetic T cell-costimulatory receptor peptides fold into a polyproline type II helical structure commonly seen in regions of globular proteins involved in transient protein-protein interactions. The binding determinants of CD28 can be transferred onto a short peptide mimic of its ligand-binding region. The CD28 peptide mimics effectively block the expansion of encephalitogenic T cells in vitro suggesting the potential usefulness of the peptides for the treatment of autoimmune disease conditions requiring down-regulation of T cell responses.

L110 ANSWER 2 OF 84 MEDLINE on STN
ACCESSION NUMBER: 2000020192 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10550750
TITLE: IFN-tau inhibits IgE production in a murine model of

allergy and in an IgE-producing human myeloma cell line.
AUTHOR: Mujtaba M G; Villarete L; Johnson H M
CORPORATE SOURCE: Department of Microbiology and Cell Science, University of Florida, Gainesville 32611, USA.
CONTRACT NUMBER: CA69959 (NCI)
R37 AI25904 (NIAID)
SOURCE: Journal of allergy and clinical immunology, (1999 Nov) 104 (5) 1037-44.
Journal code: 1275002. ISSN: 0091-6749.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199912
ENTRY DATE: Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991222
ED Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991222
AB BACKGROUND: IFN-tau, a type I IFN, is an antiviral, immunomodulating, and antiproliferative agent similar to IFN-alpha and IFN-beta, but IFN-tau lacks the toxicity associated with high concentrations of these IFNs in tissue culture and in animal studies. We have previously shown that IFN-tau inhibits antibody production in a murine model of an autoimmune disease. OBJECTIVE: We investigate the effectiveness of ovine IFN-tau and other type I IFNs in suppressing the development of allergic sensitization in a murine model of allergy by using ovalbumin (OVA) antigen as an allergen and in suppressing IgE production by using a human IgE-producing myeloma cell line.Methods and Results: Mice that were treated with IFN-tau in vivo before and after intraperitoneal immunization with aluminum hydroxide-precipitated OVA had significantly lower OVA-specific IgE levels than the PBS-treated group. IFN-tau-treated mice had reduced inflammatory cell infiltration into the lung tissue. Furthermore, in vitro IFN-tau treatment of splenocytes taken from OVA-immunized mice suppressed OVA-induced proliferation. Also, treatment of the IgE-producing human myeloma cell line U266BL with IFN-tau-reduced IgE production and inhibited cell proliferation compared with media controls. Similar suppression of proliferation and inhibition of IgE production was seen with other type I IFNs, as well as a humanized IFN-tau/IFN-alphaD chimeric that consists of residues 1 to 27 of the ovine IFN-tau and residues 28 to 166 of the human IFN-alphaD. The chimeric was not toxic to human peripheral white blood cells at concentrations as high as 10(5) U/mL, whereas human IFN-alphaD was toxic at 10(3) U/mL. CONCLUSION: These data suggest that IFNs may be useful in preventing allergic sensitization by suppressing the production of allergen-specific IgE antibodies without toxic side effects.

L110 ANSWER 3 OF 84 MEDLINE on STN
ACCESSION NUMBER: 1998031776 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9366448
TITLE: Prevention of immunotoxin-induced immunogenicity by coadministration with CTLA4Ig enhances antitumor efficacy.
AUTHOR: Siegall C B; Haggerty H G; Warner G L; Chace D; Mixan B; Linsley P S; Davidson T
CORPORATE SOURCE: Molecular Immunology Department, Bristol-Myers Squibb Pharmaceutical Research Institute, Syracuse, NY 13057, USA.. Clay_B._Siegall@ccmail.bms.com
SOURCE: Journal of immunology (Baltimore, Md. : 1950), (1997 Nov 15) 159 (10) 5168-73.

Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199711
ENTRY DATE: Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971125
ED Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971125
AB Immunotoxins have shown promise as antitumor agents in clinical trials. However, they have not become part of standard cancer therapy because of factors that include their inherent immunogenicity, which limits the duration of therapy. To address this issue, we evaluated in preclinical models the concomitant use of the immunosuppressive agent CTLA4Ig and BR96 sFv-PE40, a single-chain immunotoxin that binds to carcinoma cells expressing Le(y). Cotreatment with CTLA4Ig, an inhibitor of the CD28/CTLA4-CD80/CD86 costimulation pathway, blocked the production of Abs against BR96 sFv-PE40 in immunocompetent rodents and **dogs**. It also blocked hypersensitivity reactions in rats carrying colon carcinoma allografts during a second course of BR96 sFv-PE40 therapy, and the cotreatment with CTLA4Ig resulted in enhanced antitumor activity. Cotreatment with CTLA4Ig also prevented hypersensitivity reactions induced by repeat dosing of BR96 sFv-PE40 (q3dx5) in **dogs**. The production of anti-BR96-sFv-PE40 Abs was decreased in CTLA4Ig-cotreated rodents and **dogs** resulting in increased plasma levels of BR96 sFv-PE40 relative to non-CTLA4Ig-cotreated animals. These data show that cotreatment of immunotoxins with CTLA4Ig, by inhibiting the production of anti-immunotoxin Abs, can extend the duration of BR96 sFv-PE40 therapy to give greater exposure, reduced toxicities, and increased efficacy.

L110 ANSWER 4 OF 84 MEDLINE on STN
ACCESSION NUMBER: 97472543 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9331502
TITLE: CTLA4-Ig prolongs survival of microencapsulated neonatal porcine islet xenografts in diabetic NOD mice.
AUTHOR: Weber C J; Hagler M K; Chryssochoos J T; Kapp J A; Korbutt G S; Rajotte R V; Linsley P S
CORPORATE SOURCE: Department of Surgery, Bristol Myers-Squibb, Seattle, WA, USA.
CONTRACT NUMBER: DK R01-39088 (NIDDK)
SOURCE: Cell transplantation, (1997 Sep-Oct) 6 (5) 505-8.
Journal code: 9208854. ISSN: 0963-6897.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199711
ENTRY DATE: Entered STN: 19971224
Last Updated on STN: 19990129
Entered Medline: 19971114
ED Entered STN: 19971224
Last Updated on STN: 19990129
Entered Medline: 19971114

L110 ANSWER 5 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2004:306417 HCAPLUS

DOCUMENT NUMBER: 140:337893
TITLE: Antagonists or antibodies to A33 antigens for diagnosis and therapy of immune diseases, inflammations and **cancers**
INVENTOR(S): Ashkenazi, Avi J.; Fong, Sherman; Goddard, Audrey; Gurney, Austin L.; Napier, Mary A.; Tumas, Daniel; Van Lookeren, Menno; Wood, William I.
PATENT ASSIGNEE(S): Genentech, Inc., USA
SOURCE: PCT Int. Appl., 230 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 119
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004031105	A2	20040415	WO 2003-US31207	20031001
W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, EG, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
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US 2002058309	A1	20020516	US 2001-866028	20010525 <--
US 6642360	B2	20031104		
JP 2004520811	T2	20040715	JP 2002-522282	20010530
AU 758921	B2	20030403	AU 2001-57764	20010801 <--
AU 759004	B2	20030403	AU 2001-57765	20010801 <--
JP 2004520810	T2	20040715	JP 2002-522275	20010823
US 2003207803	A1	20031106	US 2001-143026	20011019 <--
US 2003170254	A1	20030911	US 2001-17191	20011024
US 2003199021	A1	20031023	US 2001-13924	20011025 <--
EP 1397383	A2	20040317	EP 2001-990229	20011213
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AU 772723	B2	20040506	AU 2002-14769	20020201 <--
AU 772734	B2	20040506	AU 2002-14771	20020201 <--
WO 2002101069	A2	20021219	WO 2002-US10513	20020403
WO 2002101069	A3	20030904		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1402260	A2	20040331	EP 2002-731246	20020403
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

US 2003148438	A1	20030807	US 2002-145821	20020514	
US 2003170788	A1	20030911	US 2002-145634	20020514	
US 2003166084	A1	20030904	US 2002-146793	20020515	
US 2003134380	A1	20030717	US 2002-147509	20020516	<--
US 2003180875	A1	20030925	US 2002-147505	20020517	<--
US 2003199027	A1	20031023	US 2002-152396	20020520	<--
US 2003068695	A1	20030410	US 2002-192012	20020709	
US 2003068696	A1	20030410	US 2002-192014	20020709	
US 2003049743	A1	20030313	US 2002-194394	20020711	
US 2003049745	A1	20030313	US 2002-194485	20020711	
US 2003064446	A1	20030403	US 2002-194460	20020711	
US 2003153037	A1	20030814	US 2002-194457	20020711	
US 2003059879	A1	20030327	US 2002-194456	20020712	
US 2003064448	A1	20030403	US 2002-194484	20020712	
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US 2003063112	A1	20030403	US 2002-195896	20020715	
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US 2003071834	A1	20030417	US 2002-195898	20020715	
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US 2003065159	A1	20030403	US 2002-196757	20020716	
US 2003068710	A1	20030410	US 2002-196761	20020716	
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US 2003064463	A1	20030403	US 2002-206922	20020726	
US 2003068756	A1	20030410	US 2002-206912	20020726	
US 2003068759	A1	20030410	US 2002-206920	20020726	
US 2003068760	A1	20030410	US 2002-206921	20020726	
US 2003073183	A1	20030417	US 2002-206917	20020726	
US 2003096359	A1	20030522	US 2002-205910	20020726	
US 2004048334	A1	20040311	US 2002-205890	20020726	
US 2003068765	A1	20030410	US 2002-207916	20020729	
US 2003068766	A1	20030410	US 2002-207917	20020729	
US 2003068769	A1	20030410	US 2002-207920	20020729	
US 2003068773	A1	20030410	US 2002-208023	20020729	
US 2003068774	A1	20030410	US 2002-208026	20020729	
US 2003073184	A1	20030417	US 2002-207923	20020729	
US 2003073185	A1	20030417	US 2002-207924	20020729	
US 2003215912	A1	20031120	US 2002-207915	20020729	
US 2004048335	A1	20040311	US 2002-208024	20020729	
US 2003171568	A1	20030911	US 2002-265542	20021003	<--
US 2003120056	A1	20030626	US 2002-289498	20021105	
US 2003144498	A1	20030731	US 2002-289527	20021105	
US 2003224984	A1	20031204	US 2002-305654	20021126	
US 2003199044	A1	20031023	US 2003-410552	20030408	<--
US 2004120957	A1	20040624	US 2003-633008	20030731	<--

PRIORITY APPLN. INFO.:

US 2002-265542	A	20021003	
US 2003-633008	A	20030731	
US 1997-63564P	P	19971028	<--
US 1997-63870P	P	19971031	<--
US 1998-82704P	P	19980422	<--
US 1998-85339P	A1	19980513	<--
US 1998-87106P	P	19980528	<--
US 1998-88655P	P	19980609	<--

US	1998-89947P	P	19980619 <--
US	1998-94651P	A1	19980730 <--
US	1998-97974P	P	19980826 <--
AU	1998-93881	A3	19980914 <--
AU	1998-93178	A3	19981002 <--
WO	1998-US24855	A1	19981120 <--
US	1998-216021	B1	19981216 <--
US	1998-218517	B1	19981222 <--
US	1999-254311	A1	19990303 <--
US	1999-254465	A2	19990305 <--
US	1999-131293P	P	19990427 <--
US	1999-380139	A1	19990825 <--
US	1999-920594	A	19990908 <--
US	1999-921090	A	19990915 <--
US	1999-99309	A	19991220 <--
US	2000-441400	A	20000222
WO	2000-US4414	A2	20000222
WO	2000-US6471	W	20000309
US	2000-198121P	P	20000418
US	2000-198585P	P	20000418
US	2000-199397P	P	20000425
US	2000-199550P	P	20000425
US	2000-201516P	P	20000503
US	2000-204675P	P	20000517
US	2000-232887P	P	20000915
US	2000-690189	A3	20001016
WO	2001-US17443	W	20010530
US	2001-880457	A	20010612
WO	2001-US26626	W	20010823
US	2001-953499	A2	20010914
US	2001-2796	A	20011115
WO	2001-US48938	W	20011213
US	2002-52586	A1	20020115
WO	2002-US10513	W	20020403

ED Entered STN: 15 Apr 2004

AB The present invention relates to compns. and methods of treating and diagnosing disorders characterized the by the presence of antigens associated with inflammatory diseases and/or **cancer**. Antigens associated with inflammation and inflammatory diseases and **cancer** are identified for use as diagnostic markers and in the treatment of the disease. The A33 antigens STIgMA, PRO301, PRO362, PRO245, and PRO1868 are identified. Genes for the antigens were identified by screening proprietary databases for secreted proteins. Primers derived from consensus sequences were used to cloned cDNAs for the proteins. Two of the proteins, PRO301 and PRO245, inhibited vascular endothelial growth factor stimulation of endothelial cell proliferation and stimulate T cell proliferation. They are also proinflammatory and stimulate the infiltration of luekocytes into guinea **pig** skin.

L110 ANSWER 6 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2004:550641 HCAPLUS
DOCUMENT NUMBER: 141:83636
TITLE: Protein and cDNA sequences of G protein-coupled receptor proteins (LPLRF and 0T7T022) from human, bovine, rat and mouse, their derived peptides as GPCR againsts, and diagnostic and therapeutic use thereof
INVENTOR(S): Watanabe, Takuya; Kikuchi, Kuniko; Terao, Yasuko; Shintani, Yasushi; Hinuma, Shuji; Fukusumi, Shuji; Fujii, Ryo; Hosoya, Masaki; Kitada, Chieko

PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 107 pp., Cont.-in-part of U.S. Ser. No. 831,758, abandoned.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004132073	A1	20040708	US 2003-719587	20031121 <--
WO 2000029441	A1	20000525	WO 1999-JP6283	19991111 <--

W: AE, AL, AM, AU, AZ, BA, BB, BG, BR, BY, CA, CN, CR, CU, CZ, DM, EE, GD, GE, HR, HU, ID, IL, IN, IS, JP, KG, KR, KZ, LC, LK, LR, LT, LV, MA, MD, MG, MK, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, US, UZ, VN, YU, ZA, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:			
JP 1998-323759	A	19981113 <--	
JP 1999-60030	A	19990308 <--	
JP 1999-106812	A	19990414 <--	
JP 1999-166672	A	19990614 <--	
JP 1999-221640	A	19990804 <--	
JP 1999-259818	A	19990914 <--	
WO 1999-JP6283	W	19991111 <--	
US 2001-831758	B2	20010511	

ED Entered STN: 09 Jul 2004
AB The present inventions provide protein and cDNA sequences of G protein-coupled receptor proteins (termed LPLRF) from human, bovine, rat and mouse; and related G protein-coupled receptor 0T7T022L and 0T7T022 isolated from rat and human resp. The LPLRF cDNAs are isolated from physiol. active peptide cDNA from corresponding animal brain cDNA and their derived peptides are confirmed to react to monoclonal antibody 1F3 against rat RFRP-1, which is a sequence homolog of prolactin-releasing peptide. In particular embodiments, various LPLRF amide peptides derived from human LPLRF by amidating their C-terminal carboxy groups, are shown to function as agonists of human GPCR 0T7T022 in CHO cells. The 0T7T022 receptor-mediated cAMP production is inhibited by pertussis toxin in the presence of RFRP derived peptide. The polypeptides in the present invention possess the effects of promoting and inhibiting the secretion of prolactin, and are thus useful as drugs for the prevention and treatment of various diseases, in terms of prolactin secretion stimulants, which are associated with the secretion of prolactin, such as hypoovarianism, spermatic underdevelopment, menopausal symptoms, hypothyroidism, etc. The polypeptides are useful as drugs for the prevention and treatment of various diseases, in terms of prolactin secretion inhibitors, which are associated with the secretion of prolactin, such as pituitary **tumor**, diencephalon **tumor**, menstrual disorder, autoimmune diseases, prolactinoma, sterility, impotence, amenorrhea, lactorrhea, acromegaly, Chiari-Frommel syndrome, Argonz-del Castilo syndrome, Forbes-Albright syndrome, **lymphoma**, Sheehan's syndrome, spermatogenesis disorder, etc.

L110 ANSWER 7 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2004:59558 HCAPLUS
DOCUMENT NUMBER: 140:127193

TITLE: Vaccines comprising aggregating protein epitopes and antibodies for treating a plaque-forming neurological or CNS disease
INVENTOR(S): Solomon, Beka; Frenkel, Dan
PATENT ASSIGNEE(S): Ramot At Tel-Aviv University Ltd., Israel
SOURCE: U.S. Pat. Appl. Publ., 68 pp., Cont.-in-part of U.S. Ser. No. 162,889.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004013647	A1	20040122	US 2003-384788	20030311 <--
US 6703015	B1	20040309	US 1999-473653	19991229 <--
WO 2001018169	A2	20010315	WO 2000-IL518	20000831 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2002052311	A1	20020502	US 2001-808037	20010315 <--
US 2003077252	A1	20030424	US 2002-162889	20020606 <--
US 2004052766	A1	20040318	US 2003-618856	20030715 <--

PRIORITY APPLN. INFO.:

US 1999-152417P	P	19990903 <--
US 1999-473653	B2	19991229 <--
US 2000-629971	B2	20000731
WO 2000-IL518	W	20000831
US 2001-808037	B2	20010315
US 2001-830954	A2	20010807
US 2002-371735P	P	20020412
US 2002-162889	A2	20020606

ED Entered STN: 23 Jan 2004
AB A method of immunizing against plaque forming diseases using display technol. is provided. The method utilize novel agents, or pharmaceutical compns. for vaccination against plaque forming diseases which rely upon presentation of an antigen or epitope on a display vehicle. The method further includes agents, or pharmaceutical compns. for vaccination against plaque forming diseases, which rely upon presentation of an antibody, or an active portion thereof, on a display vehicle. Whether antigens or antibodies are employed, disaggregation of plaques results from the immunization. The methods of the present invention also generally relates to treating and/or diagnosing neurol. diseases and disorders of the central nervous, regardless of whether the disease or disorder is plaque-forming or non-plaque forming.

L110 ANSWER 8 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:417777 HCAPLUS
DOCUMENT NUMBER: 139:5653
TITLE: Histone-derived peptides for diagnosis and therapy of autoimmune disease
INVENTOR(S): Zeppezauer, Michael; Schoenberger, Arno; Cebecauer, Ladislav

PATENT ASSIGNEE(S): Symbiotec Gesellschaft zur Erforschung und Entwicklung auf dem Gebiete der Biotechnology M.b.h., Germany
SOURCE: PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003044054	A2	20030530	WO 2002-EP12955	20021119
WO 2003044054	A3	20040304		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2003144473	A1	20030731	US 2001-988165	20011119 <--
PRIORITY APPLN. INFO.:			US 2001-988165	A 20011119
			US 1992-946180	A2 19920916 <--

ED Entered STN: 01 Jun 2003
AB The authors disclose peptides which react with autoantibodies in the body fluids of patients, who are suffering from autoimmune diseases, in particular diseases of the rheumatic group as systemic lupus erythematosus (SLE), rheumatoid arthritis or systemic sclerosis. The antigenic peptides are derived from the C-terminus of histone H1 (bovine or human sub-types H1.1, H1.2, H1.3, H1.4, H1.5 and H1.a) and the N-termini of histone H2B with the sequence section 1-35 and 36-76 and are capable of cross-reactions with the autoantibodies (anti-histone-antibodies). The invention furthermore provides ways of forming monoclonal antibodies and anti-idiotypic antibodies, which are directed against autoantibodies. The diagnosis of autoimmune peptides and diseases is possible in accordance with the invention with a high degree of certainty and the monoclonal antibodies directed against the autoantibodies are suitable for the production of medicaments for the therapy of said diseases.

L110 ANSWER 9 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:971282 HCAPLUS
DOCUMENT NUMBER: 140:26899
TITLE: Method and compositions for stimulation of an immune response to CD20 using a xenogeneic CD20 antigen
INVENTOR(S): Palomba, Maria Lia; Houghton, Alan; Wolchok, Jedd; Scheinberg, David A.; Roberts, Wendy K.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 17 pp., Cont.-in-part of U.S. Ser. No. 627,694.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003228326	A1	20031211	US 2002-285874	20021031 <--
WO 9825574	A2	19980618	WO 1997-US22669	19971210 <--
WO 9825574	A3	19980903		
W: CA, JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 6328969	B1	20011211	US 1999-308697	19990521 <--
PRIORITY APPLN. INFO.:			US 1999-308697 A2	19970217 <--
			US 1997-36419P P	19970218 <--
			WO 1997-US22669 W	19971210 <--
			US 2000-627694 A2	20000728
			US 1996-32535P P	19961210 <--

ED Entered STN: 12 Dec 2003

AB Tolerance of the immune system for endogenous CD20 can be overcome and an immune response stimulated by administration of xenogeneic or xenoexpressed CD20 antigen. For example, mouse CD20, or antigenically-effective portions thereof, can be used to stimulate an immune response to the corresponding differentiation antigen in a human subject. Administration of xenogeneic antigens in accordance with the invention results in an effective immunity against CD20 expressed by the **cancer** in the treated individual, thus providing a therapeutic approach to the treatment of **lymphomas** and **leukemia** expressing CD20. For production of a recombinant mouse CD20 fusion protein (recCD20) the inventors used the baculovirus expression system to obtain a partially purified recCD20 for the use as xenoexpressed CD20.

L110 ANSWER 10 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:590868 HCAPLUS
DOCUMENT NUMBER: 139:148460
TITLE: Peptides for the production of preparations for the diagnosis and therapy of autoimmune diseases
INVENTOR(S): Zeppezauer, Michael; Schonberger, Arno; Cebecauer, Ladislav
PATENT ASSIGNEE(S): Germany
SOURCE: U.S. Pat. Appl. Publ., 7 pp., Cont.-in-part of U.S. Ser. No. 946,180.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003144473	A1	20030731	US 2001-988165	20011119 <--
US 6369203	B1	20020409	US 1992-946180	19920916
WO 2003044054	A2	20030530	WO 2002-EP12955	20021119
WO 2003044054	A3	20040304		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR,				

NE, SN, TD, TG
PRIORITY APPLN. INFO.: US 1992-946180 A2 19920916 <--
US 2001-988165 A 20011119

ED Entered STN: 01 Aug 2003
AB Peptides are proposed with antigenic or immunogenic determinants, resulting from autoantibodies in the body fluids of patients with autoimmune diseases, in particular diseases of the rheumatic group such as systemic lupus erythematosus (SLE), rheumatoid arthritis, or systemic sclerosis. Peptides are preferably from the C terminus of bovine histone H1 with the sequence (187-211) or corresponding human histone H1-peptides of the sub-types H1.1, H1.2, H1.3, H1.4, H1.5, and H1.a, and the N termini of histone H2B with the sequences (1-35) and (36-76), which are capable of cross reactions with the autoantibodies (anti-histone-antibodies). The invention furthermore provides ways of preparing monoclonal antibodies and antiidiotypic antibodies, which are directed against autoantibodies. The diagnosis of autoimmune diseases is possible in accordance with the invention with a high degree of certainty and the monoclonal antibodies directed against the autoantibodies are suitable for the production of medicaments for the therapy of said diseases.

L110 ANSWER 11 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:512078 HCAPLUS
DOCUMENT NUMBER: 139:67774
TITLE: Immunization of animals by topical applications of a Salmonella-based vector
INVENTOR(S): Tang, De-chu C.; Marks, Donald H.; Curiel, David T.; Shi, Zhongkai; Van Kampen, Kent Rigby
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 42 pp., Cont.-in-part of U.S. Ser. No. 563,826.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003125278	A1	20030703	US 2002-52323	20020118 <--
WO 9908713	A1	19990225	WO 1998-US16739	19980813 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6706693	B1	20040316	US 2000-402527	20000103 <--
US 6716823	B1	20040406	US 2000-533149	20000323 <--
US 6348450	B1	20020219	US 2000-563826	20000503 <--
ZA 2001009348	A	20030522	ZA 2001-9348	20011113 <--
US 2003045492	A1	20030306	US 2002-116963	20020405 <--
US 2004009936	A1	20040115	US 2003-346021	20030116 <--
WO 2003070920	A1	20030828	WO 2003-US1599	20030117
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,				

PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ,
UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD,
RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:	US 1997-55520P	P	19970813 <--
	US 1998-75113P	P	19980211 <--
	WO 1998-US16739	W	19980813 <--
	US 1999-132216P	P	19990503 <--
	US 2000-402527	A2	20000103
	US 2000-533149	A2	20000323
	US 2000-563826	A2	20000503
	US 2002-52323	A2	20020118
	US 2002-116963	A2	20020405

ED Entered STN: 04 Jul 2003

AB The present invention relates to techniques of skin-targeted non-invasive gene delivery to elicit immune responses and uses thereof. The invention further relates to methods of non-invasive genetic immunization in an animal and/or methods of inducing a systemic immune or therapeutic response in an animal following topical application of vectors, products therefrom and uses for the methods and products therefrom. The methods can include contacting skin of the animal with a vector in an amount effective to induce the systemic immune or therapeutic response in the animal as well as such a method further including disposing the vector in and/or on the delivery device. The vector can be gram neg. bacteria, preferably Salmonella and most preferably Salmonella typhimurium. The topical vaccine can be used to induce immune responses to **cancer** or infectious pathogens.

L110 ANSWER 12 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER:	2003:511939 HCAPLUS
DOCUMENT NUMBER:	139:80271
TITLE:	Human breast **cancer** marker protein PDEBC, its protein and cDNA sequences, and use thereof in drug screening and diagnosis
INVENTOR(S):	Stuart, Susan G.; Streeter, David G.
PATENT ASSIGNEE(S):	USA
SOURCE:	U.S. Pat. Appl. Publ., 31 pp., Cont.-in-part of U.S. 6,368,794. CODEN: USXXCO
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	4

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003124543	A1	20030703	US 2002-79111	20020220 <--
US 6368794	B1	20020409	US 1999-232160	19990115

PRIORITY APPLN. INFO.:	US 1999-232160	A2	19990115 <--

ED Entered STN: 04 Jul 2003

AB The invention provides a cDNA which encodes a protein differentially expressed in breast **cancer**. Specifically, the protein and cDNA sequences for a human breast **cancer** marker protein PDEBC are provided. An antigenic epitope of PDEBC (amino acids 30-50) is also provided for antibody preparation It also provides for the use of the cDNA, fragments, complements, and variants thereof and of the encoded protein,

portions thereof and antibodies thereto for diagnosis and treatment of **cancer**, particularly a breast **cancer**. The invention addnl. provides expression vectors and host cells for the production of the protein and a transgenic model system. Furthermore, three PDEBC related cDNA fragments from rat and **dog** are also provided.

L110 ANSWER 13 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:435072 HCAPLUS
DOCUMENT NUMBER: 139:21017
TITLE: Prostate-associated protease HUPAP, cDNA and antibodies for prognosis, diagnosis and treatment of prostate **cancer**
INVENTOR(S): Spancake, Kimberly M.; Bandman, Olga; Lal, Preeti G.
PATENT ASSIGNEE(S): Incyte Genomics, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 42 pp., Cont.-in-part of U.S. Ser. No. 988,975.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003103981	A1	20030605	US 2002-235699	20020904 <--
US 6043033	A	20000328	US 1997-807151	19970227
US 6350448	B1	20020226	US 2000-478957	20000107 <--
US 2002119531	A1	20020829	US 2001-988975	20011119 <--
PRIORITY APPLN. INFO.:			US 1997-807151	A3 19970227 <--
			US 2000-478957	A2 20000107
			US 2001-988975	A2 20011119

ED Entered STN: 06 Jun 2003
AB The invention provides a cDNA which encodes a human prostate-associated protease, or kallikrein designated as HUPAP, differentially expressed in prostate **cancer**. It also provides for the use of the cDNA, fragments, complements, and variants thereof and of the encoded protein, portions thereof and antibodies thereto for diagnosis and treatment of prostate **cancer**. The invention addnl. provides expression vectors and host cells for the production of the protein and a transgenic model system.

L110 ANSWER 14 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:413988 HCAPLUS
DOCUMENT NUMBER: 139:5158
TITLE: Prostate **cancer** marker PCADM-1 and related compositions, methods, and kits based on DNA macroarray proteomics platforms for **cancer** diagnosis and therapy
INVENTOR(S): Stearns, Mark; Hu, Youji; Wang, Min
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 65 pp., Cont.-in-part of U.S. Ser. No. 813,380.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

US 2003100033	A1	20030529	US 2002-140602	20020507 <--
WO 2001021828	A1	20010329	WO 2000-US25981	20000921 <--

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 2002042062	A1	20020411	US 2001-813380	20010321 <--
US 2003207339	A1	20031106	US 2002-98992	20020315 <--
WO 2002083081	A2	20021024	WO 2002-US8673	20020321
WO 2002083081	A3	20021212		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

WO 2003094844	A2	20031120	WO 2003-US14098	20030507

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:	US 1999-155865P	P	19990924 <--
	WO 2000-US25981	A1	20000921
	US 2001-813380	A2	20010321
	US 2002-98992	A2	20020315
	WO 2002-US8673	A2	20020321
	US 2002-140602	A	20020507

ED Entered STN: 30 May 2003

AB The invention relates to novel nucleic acids encoding a mammalian PCADM-1 (prostate **cancer** antigen diagnostic marker 1) gene, and proteins encoded thereby, whose expression is increased in certain diseases, disorders, or conditions, including, but not limited to, prostate **cancer**. The invention further relates to methods of detecting and treating prostate **cancer**, comprising modulating or detecting PCADM-1 expression and/or production and activity of PCADM-1 polypeptide. Further, the invention relates to novel assays for the identification of DNA-binding proteins and the double-stranded oligonucleotide sequences that specifically bind with them. Finally, the invention relates to DNAZYMs or DNA enzymes which specifically bind PCADM-1 mRNA to inhibit PCADM-1 gene expression and thereby destroy **tumor** cells and **tumor** tissue.

L110 ANSWER 15 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:355743 HCAPLUS
DOCUMENT NUMBER: 138:348690
TITLE: Therapeutic antiangiogenic compositions containing endostatin and methods of endostatin preparation
INVENTOR(S): O'Reilly, Michael S.; Folkman, M. Judah
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 35 pp., Cont. of U.S. Ser. No. 174,381.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE	
US 2003087393	A1	20030508	US 2002-232316		20020903	<--
US 6746865	B1	20040608	US 1998-174381		19981016	<--
PRIORITY APPLN. INFO.:			US 1998-174381	A1	19981016	<--
			US 1995-5835P	P	19951023	<--
			US 1996-23070P	P	19960802	<--
			US 1996-26263P	P	19960917	<--
			US 1996-740168	A1	19961022	<--

ED Entered STN: 09 May 2003
AB An inhibitor of endothelial cell proliferation, capable of inhibiting angiogenesis and causing **tumor** regression, that is approx. 20 kDa and corresponds to a C-terminal fragment of collagen type XVIII, and methods of treating angiogenesis-related disease are claimed. A preferred endothelial cell proliferation inhibitor of the invention is a protein having the above-described characteristics, and which can be isolated and purified from the murine hemangioendothelioma cell line EOMA. This inhibitory protein has been named endostatin. A composition comprising, angiostatin combined with the endostatin of the invention and a method of making endostatin protein are also claimed.

L110 ANSWER 16 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:222247 HCAPLUS
DOCUMENT NUMBER: 138:253105
TITLE: **Tumor** suppressor gene and protein EPLIN (epithelial protein lost in neoplasm) playing a role in regulation of cell proliferation and their uses
INVENTOR(S): Chang, David D.; Maul, Raymond S.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 51 pp., Cont.-in-part of U.S. Ser. No. 658,400.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE	
US 2003054417	A1	20030320	US 2001-783732		20010213	<--
PRIORITY APPLN. INFO.:			US 1999-153024P	P	19990908	<--
			US 2000-658400	A2	20000908	

ED Entered STN: 21 Mar 2003
AB A novel **tumor** suppressor gene and the protein it encodes: EPLIN

(epithelial protein lost in neoplasm) that may be of use in the diagnosis and treatment of **cancer** are described. Also included is a method for detecting a cell proliferative disorder associated with EPLIN. EPLIN is a marker that can be used diagnostically, prognostically and therapeutically over the course of cell proliferative disorders associated with EPLIN.

L110 ANSWER 17 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:174471 HCAPLUS
DOCUMENT NUMBER: 138:203673
TITLE: Vaccination by topical application of recombinant vectors
INVENTOR(S): Tang, De-chu C.; Shi, Zhongkai; Rigby Van Kampen, Kent
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 44 pp., Cont.-in-part of U.S. Ser. No. 533,149.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003045492	A1	20030306	US 2002-116963	20020405 <--
WO 9908713	A1	19990225	WO 1998-US16739	19980813 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
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US 6706693	B1	20040316	US 2000-402527	20000103 <--
US 6716823	B1	20040406	US 2000-533149	20000323 <--
US 6348450	B1	20020219	US 2000-563826	20000503 <--
ZA 2001009348	A	20030522	ZA 2001-9348	20011113 <--
US 2003125278	A1	20030703	US 2002-52323	20020118 <--
US 2004009936	A1	20040115	US 2003-346021	20030116 <--
WO 2003070920	A1	20030828	WO 2003-US1599	20030117
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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.:		
US 1997-55520P	P	19970813 <--
US 1998-75113P	P	19980211 <--
WO 1998-US16739	W	19980813 <--
US 1999-132216P	P	19990503 <--
US 2000-402527	A2	20000103
US 2000-533149	A2	20000323
US 2000-533149	A2	20000323

L110 ANSWER 23 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:276421 HCAPLUS
DOCUMENT NUMBER: 136:278150
TITLE: Immunotherapy of **malignant** and autoimmune disorders in domestic animals using **naked** antibodies, **immunoconjugates** and fusion proteins
INVENTOR(S): Goldenberg, David M.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 17 pp., Cont.-in-part of U.S. 6,134,982.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE	
US 2002041847	A1	20020411	US 2001-921290		20010803	<--
US 6306393	B1	20011023	US 1999-307816		19990510	<--
PRIORITY APPLN. INFO.:			US 1998-38995	A2	19980312	<--
			US 1999-307816	A2	19990510	<--
			US 1997-41506P	P	19970324	<--

ED Entered STN: 12 Apr 2002
AB B-cell, T-cell, **myeloid-cell**, **mast-cell**, and **plasma-cell** disorders are significant contributors to illness and mortality in domestic animals, especially in companion animals such as **dogs** and **cats**. These disorders include both autoimmune disorders and **malignancies**, such as the B-cell subtype of non-Hodgkin's **lymphoma**, acute and chronic lymphocytic or myeloid **leukemias**, multiple **myeloma**, and **mastocytomas**. Antibody components that bind with B-cell or T-cell antigens or epitopes, as well as antigens or epitopes of myeloid, plasma and **mast cells** provide an effective means to treat these disorders in domestic animals. The immunotherapy uses **naked** antibodies, **immunoconjugates** and fusion proteins, alone or in combination with standard therapeutic regimens.

L110 ANSWER 24 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:185718 HCAPLUS
DOCUMENT NUMBER: 136:261822
TITLE: Humanized anti-VEGF antibodies for diagnosis and treatment of neovascularization and neoplasm
INVENTOR(S): Baca, Manuel; Wells, James A.; Presta, Leonard G.; Lowman, Henry B.; Chen, Yvonne Man-yee
PATENT ASSIGNEE(S): Australia
SOURCE: U.S. Pat. Appl. Publ., 47 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
US 2002032315	A1	20020314	US 1998-56160	19980406	<--
US 2003190317	A1	20031009	US 2002-234671	20020903	<--

PRIORITY APPLN. INFO.: US 1997-54856P P 19970806 <--
US 1997-126446P P 19970407 <--
US 1998-56160 B1 19980406 <--

ED Entered STN: 15 Mar 2002
AB Humanized and variant anti-VEGF antibodies and various uses therefor are disclosed. The anti-VEGF antibodies have strong binding affinities for VEGF; inhibit VEGF-induced proliferation of endothelial cells in vitro; and inhibit angiogenesis and **tumor** growth in vivo.

L110 ANSWER 25 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:11026 HCAPLUS
DOCUMENT NUMBER: 136:80831
TITLE: Cytoplasmic transfer to de-differentiate recipient cells
INVENTOR(S): Chapman, Karen B.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 9 pp., Cont.-in-part of Appl. No. PCT/US00/18063.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002001842	A1	20020103	US 2000-736268	20001215 <--
WO 2001000650	A1	20010104	WO 2000-US18063	20000630 <--

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-141250P P 19990630 <--
WO 2000-US18063 A2 20000630

ED Entered STN: 04 Jan 2002
AB Methods for de-differentiating or altering the life-span of desired "recipient" cells, e.g., human somatic cells, by the introduction of cytoplasm from a more primitive, less differentiated cell type, e.g., oocyte or blastomere are provided. These methods can be used to produce embryonic stem cells and to increase the efficiency of gene therapy by allowing for desired cells to be subjected to multiple genetic modifications without becoming senescent. Such cytoplasm may be fractionated and/or subjected to subtractive hybridization and the active materials (sufficient for de-differentiation) identified and produced by recombinant methods.

L110 ANSWER 26 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:711282 HCAPLUS
DOCUMENT NUMBER: 137:246516
TITLE: Recombinant Newcastle disease virus RNA expression systems for expressing foreign antigen as vaccine against HIV, **cancer**, autoimmune disease, infection or allergy
INVENTOR(S): Garcia-Sastre, Adolfo; Palese, Peter

PATENT ASSIGNEE(S): Mount Sinai School of Medicine of New York University, USA
SOURCE: U.S., 27 pp., Cont.-in-part of U.S. 6,146,642.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6451323	B1	20020917	US 2000-576567	20000522 <--
US 6146642	A	20001114	US 1998-152845	19980914
ZA 2001002546	A	20020902	ZA 2001-2546	20010328 <--
US 2003078410	A1	20030424	US 2002-245644	20020917 <--
PRIORITY APPLN. INFO.:			US 1998-152845 A2	19980914 <--
			WO 1999-US21081 W	19990914 <--
			US 2000-576567 A1	20000522

ED Entered STN: 19 Sep 2002
AB This invention relates to genetically engineered Newcastle disease viruses and viral vectors which express heterologous genes or mutated Newcastle disease viral genes or a combination of viral genes derived from different strains of Newcastle disease virus. The invention relates to the construction and use of recombinant neg. strand NDV viral RNA templates which may be used with viral RNA-directed RNA polymerase to express heterologous gene products in appropriate host cells and/or to rescue the heterologous gene in virus particles. In a specific embodiment of the invention, the heterologous gene product is a peptide or protein derived from the genome of a human immunodeficiency virus. The RNA templates of the present invention may be prepared by transcription of appropriate DNA sequences using any DNA-directed RNA polymerase such as bacteriophage T7, T3, SP6 polymerase, or eukaryotic polymerase I.
REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L110 ANSWER 27 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:590280 HCAPLUS
DOCUMENT NUMBER: 137:139374
TITLE: Interleukin 10 with immunosuppressive but not immunostimulatory side-effects for treating inflammatory diseases and in transplantation
INVENTOR(S): Bromberg, Jonathan S.; Ding, Yaozhong; Qin, Lihui
PATENT ASSIGNEE(S): The Regents of the University of Michigan, USA
SOURCE: U.S., 42 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6428985	B1	20020806	US 1999-452624	19991130 <--
PRIORITY APPLN. INFO.:			US 1998-110601P P	19981202 <--

ED Entered STN: 08 Aug 2002
AB Disclosed is the surprising discovery that a single amino acid provides the demarcation between the immunosuppressive and immunostimulatory properties of the cytokine, IL-10. The present invention thus provides mammalian and human IL-10 genes and polypeptides that have

immunosuppressive properties, without immunostimulatory side-effects. Also provided are various methods of using the new IL-10 constructs, both in vitro and in vivo, particularly in sole or combination therapies involving immunosuppression, such as in the treatment of inflammatory diseases and disorders, and in transplantation.

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L110 ANSWER 28 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:380648 HCAPLUS
DOCUMENT NUMBER: 135:4471
TITLE: Antibodies
INVENTOR(S): Kingsman, Alan; Kingsman, Susan Mary; Bebbington, Christopher Robert; Carroll, Miles William; Ellard, Fiona Margaret; Myers, Kevin Alan
PATENT ASSIGNEE(S): Oxford Biomedica (UK) Limited, UK
SOURCE: PCT Int. Appl., 117 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001036486	A2	20010525	WO 2000-GB4317	20001113 <--
WO 2001036486	A3	20020510		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
WO 2000029428	A2	20000525	WO 1999-GB3859	19991118 <--
WO 2000029428	A3	20001109		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
GB 2371803	A1	20020807	GB 2002-12763	19991118 <--
EP 1242456	A2	20020925	EP 2000-974682	20001113 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2003515323	T2	20030507	JP 2001-538975	20001113 <--
WO 2002038612	A2	20020516	WO 2001-GB5004	20011113
WO 2002038612	A3	20030508		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA,				

UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002014150	A5	20020521	AU 2002-14150		20011113
EP 1347994	A2	20031001	EP 2001-982608		20011113
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
US 2003083290	A1	20030501	US 2002-60585		20020129 <--
US 2004131591	A1	20040708	US 2002-334235		20021230 <--
US 2004081656	A1	20040429	US 2003-416612		20031031
PRIORITY APPLN. INFO.:			WO 1999-GB3859	W	19991118 <--
			GB 2000-3527	A	20000215
			GB 2000-5071	A	20000302
			GB 1997-11579	A	19970604 <--
			GB 1997-13150	A	19970620 <--
			GB 1997-14230	A	19970704 <--
			GB 1998-25303	A	19981118 <--
			GB 1999-1739	A	19990127 <--
			GB 1999-17995	A	19990730 <--
			US 2000-445375	A2	20000321
			WO 2000-GB4317	W	20001113
			WO 2001-GB5004	W	20011113
			US 2002-60585	A2	20020129

ED Entered STN: 27 May 2001

AB The use of an ScFv Ab (ScFv Ab) capable of recognizing a disease associated mol. (DAM) in the manufacture of a medicament for the prevention and/or treatment of a disease condition associated with a DAM is described. The ScFv Ab has therapeutic, diagnostic and prognostic applications.

L110 ANSWER 29 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:380608 HCAPLUS
DOCUMENT NUMBER: 135:478
TITLE: Mammalian gonadotropin-releasing hormone (GnRH) receptor expression cassette and therapeutic uses thereof
INVENTOR(S): McArdle, Craig Alexander
PATENT ASSIGNEE(S): University of Bristol, UK
SOURCE: PCT Int. Appl., 78 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001036446	A2	20010525	WO 2000-GB4385	20001117 <--
WO 2001036446	A3	20020510		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 2001014050	A5	20010530	AU 2001-14050	20001117 <--

PRIORITY APPLN. INFO.: GB 1999-27215 A 19991117 <--
WO 2000-GB4385 W 20001117

ED Entered STN: 27 May 2001

AB The invention provides a pro-drug comprising a vector encoding a G-protein coupled receptor (GPCR). The GPCR may be modified from the naturally occurring GPCR, and can be modified in at least one of the following manner: 1. an N-terminal portion of a GPCR from which a part or all of the C-terminal tail has been truncated in order to remove one or more serine or threonine residues; 2. a GPCR in which one or more of the serine and/or threonine residues in the C-terminal tail have been mutated to prevent their phosphorylation; 3. the entire sequence of a GPCR lacking a C-terminal tail with an added C-terminal tail containing potential sites for phosphorylation and β-arrestin binding; 4. a GPCR in which one or more serine and/or threonine residues in the 3rd intracellular loop have been mutated to prevent their phosphorylation; 5. a GPCR in which the 3rd intracellular loop has been replaced with one from another GPCR; a fusion protein consisting of the entire sequence of a GPCR in tandem with the sequence of a G-protein α-subunit. A preferred GPCR for the pro-drug may be gonadotropin-releasing hormone receptor(GnRH-R) or a somatostatin receptor. The invention further provides data showing (1) that recombinant adenovirus can be used to express GnRH-Rs in hormone-dependent **cancer** cells (2) that these receptors are functional and retain pharmacol. characteristics (3) that activation of such receptors can cause a pronounced anti-proliferative effect (4) that the magnitude of this effect is dependent upon receptor number (5) that the magnitude of the effect can be limited by receptor desensitization and/or internalization and (6) that rates of desensitization and internalization of the GnRH-R are dependent upon receptor structure and can be altered by modification of receptor structure. The invention further relates to the uses of GPCR expression cassette in treatments of GPCR-associated diseases.

L110 ANSWER 30 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:360036 HCAPLUS
DOCUMENT NUMBER: 134:365710
TITLE: Modulating IL-13 activity using mutated IL-13 molecules that are antagonists or agonists of IL-13
INVENTOR(S): Puri, Raj K.; Oshima, Yasuo; Joshi, Bharat H.
PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA
SOURCE: PCT Int. Appl., 129 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001034645	A2	20010517	WO 2000-US31044	20001110 <--
WO 2001034645	A3	20020307		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 2001015993	A5	20010606	AU 2001-15993	20001110 <--

EP 1263785 A2 20021211 EP 2000-978539 20001110 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
PRIORITY APPLN. INFO.: US 1999-165236P P 19991111 <--
WO 2000-US31044 W 20001110

ED Entered STN: 18 May 2001
AB The present invention provides antagonists and agonists of IL-13 activity. The antagonists comprise a mutation in which the glutamic acid at position 13 of the IL-13 mol. is changed to a neutral or a pos. charged mol. The antagonists can be used to reduce or end symptoms in conditions, such as asthma, allergic rhinitis, atopic dermatitis, and hepatic fibrosis in schistosomiasis, in which IL-13 is an initiator, mediator, or enhancer of the abnormal state. Addnl., the antagonists can be used to slow the growth of cells of **cancers** for which IL-13 is an autocrine growth factor. Such **cancers** include renal cell **carcinoma**, Kaposi's sarcoma, and Hodgkin's disease. The agonists comprise mutated IL-13s in which one or more of the residues at positions 112, 110, 109, 92, 69, or 66 are mutated to a neutrally charged residue, or one with a charge opposite to the charge of the residue found at that position in native IL-13, provided that the residue at position 13 of the mol. is not neg. charged. The agonists can be used as more potent agents to provoke an effect provided by IL-13. In particular, the agonists can be used as reagents in the maturation of monocytes into dendritic cells, or to pretreat bone marrow stem cell donors to reduce graft vs. host disease in the recipient of the stem cells. Finally, the invention provides IL-13 receptor binding mols. with affinity for the IL-13 receptor at least about 3 times greater than that exhibited by wild-type IL-13. Also provided are methods and compns. for specifically delivering an effector mol. to a **tumor** cell by chimeric mols. comprising the effector mol. and an IL-13 receptor binding mol., and pharmaceutical compns. comprising such chimeric mols.

L110 ANSWER 31 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:338579 HCAPLUS
DOCUMENT NUMBER: 134:365705
TITLE: Antibody diversity generation
INVENTOR(S): Karrer, Erik; Bass, Steven H.; Whalen, Robert; Patten, Phillip A.
PATENT ASSIGNEE(S): Maxygen, Inc., USA
SOURCE: PCT Int. Appl., 109 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001032712	A2	20010510	WO 2000-US30247	20001101 <--
WO 2001032712	A3	20020321		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1230269 A2 20020814 EP 2000-976844 20001101 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
PRIORITY APPLN. INFO.: US 1999-163370P P 19991103 <--
US 2000-176002P P 20000112
WO 2000-US30247 W 20001101

ED Entered STN: 11 May 2001
AB Methods for improving antibodies by a variety of DNA diversification and selection procedures are provided. Improvements include increases in affinity, alterations in specificity and effector function, as well as reduced antigenicity, e.g. humanization. Libraries of recombinant antibody sequences are provided, as are cells expressing members of such libraries. Novel phage display vectors are provided. Methods for the coevolution of an antibody and its cognate antigen are provided. Coevolution is used to evolve HIV envelope proteins with increased antigenicity and broadly neutralizing antibodies that interact therewith. Methods of improving antibodies for use in the detection of biol. warfare agents are provided.

L110 ANSWER 32 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:12295 HCAPLUS
DOCUMENT NUMBER: 134:97501
TITLE: Prion protein binding proteins and uses thereof
INVENTOR(S): Cashman, Neil R.; Dodelet, Vincent; Paramithiotis, Eustache
PATENT ASSIGNEE(S): McGill University, Can.; Caprion Pharmaceuticals, Inc.
SOURCE: PCT Int. Appl., 77 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001000235	A1	20010104	WO 2000-US17927	20000629 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1210110	A1	20020605	EP 2000-943291	20000629 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP 2003531356	T2	20031021	JP 2001-505942	20000629 <--

PRIORITY APPLN. INFO.: US 1999-342426 A2 19990629 <--
WO 2000-US17927 W 20000629

ED Entered STN: 05 Jan 2001
AB In general, the invention features prion protein binding proteins (PrPBPs) and diagnostic, therapeutic, and decontamination uses thereof. The invention also features fusion protein reagents for PrPBP isolation. A PrP-alkaline phosphatase fusion protein was constructed and used to detect PrPBPs on the surfaces of mouse cell lines. These PrPBPs bound to the PrP portion of the fusion protein with high affinity, in a competitive, saturable, and conformation-dependent fashion. PrPBPs were cloned and

sequenced. Clone 6 cDNA encoded a portion of protocadherin-43.
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L110 ANSWER 33 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:2816 HCAPLUS
DOCUMENT NUMBER: 136:49362
TITLE: A human 75.2 kilodalton subunit of cleavage and polyadenylation specificity factor, protein and cDNA sequences, recombinant production and therapeutic uses
INVENTOR(S): Mao, Yumin; Xie, Yi
PATENT ASSIGNEE(S): Shanghai Borong Gene Development Co., Ltd., Peop. Rep. China
SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 25 pp.
CODEN: CNXXEV
DOCUMENT TYPE: Patent
LANGUAGE: Chinese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1303862	A	20010718	CN 1999-119814	19991022 <--

PRIORITY APPLN. INFO.: CN 1999-119814 19991022 <--
ED Entered STN: 02 Jan 2002
AB The invention relates to a human CPSF (cleavage and polyadenylation specificity factor) 75.2 kilodalton subunit. The open reading frame of the cDNA encodes a protein with 684 amino acids, and an estimated mol. weight of 75.2 kilodalton based on SDS-PAGE. The invention provides the use of polypeptide and polynucleotide in a method for treatment of various kinds of diseases, such as **cancer**, blood disease, HIV infection, immune diseases, and inflammation. The invention also relates to methods, expression vectors and host cells for recombinant production of said CPSF 75.2 kilodalton subunit. The invention also relates to agonist and antagonist of said CPSF 75.2 kilodalton subunit and uses in therapy. The protein sequence of said human CPSF 75.2 kilodalton subunit shows 99% similarity to that of **cattle** CPSF 73-kilodalton subunit.

L110 ANSWER 34 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:190949 HCAPLUS
DOCUMENT NUMBER: 132:246351
TITLE: Method of using zonula occludens toxin (Zot) or zonulin to inhibit lymphocyte proliferation in an antigen-specific manner
INVENTOR(S): Fasano, Alessio; Sztein, Marcelo B.; Lu, Ruiliang; Tanner, Michael K.
PATENT ASSIGNEE(S): University of Maryland, Baltimore, USA
SOURCE: PCT Int. Appl., 95 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000015252	A1	20000323	WO 1999-US18842	19990909 <--

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU,

CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL,
IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD,
MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK,
SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK,
ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG,
CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2342771	AA	20000323	CA 1999-2342771		19990909 <--
AU 9960190	A1	20000403	AU 1999-60190		19990909 <--
AU 754142	B2	20021107			
EP 1113813	A1	20010711	EP 1999-969032		19990909 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI					
JP 2002524531	T2	20020806	JP 2000-569836		19990909 <--
NZ 510150	A	20021025	NZ 1999-510150		19990909 <--
RU 2214271	C2	20031020	RU 2001-110101		19990909 <--
NO 2001001253	A	20010315	NO 2001-1253		20010313 <--
ZA 2001002071	A	20020225	ZA 2001-2071		20010313 <--
US 6733762	B1	20040511	US 2001-786319		20010503 <--
PRIORITY APPLN. INFO.:			US 1998-100266P	P	19980914 <--
			WO 1999-US18842	W	19990909 <--

ED Entered STN: 24 Mar 2000

AB Methods for using Zot or zonulin as an antigen-specific inhibitor of antigen-presenting cell (APC) activity and lymphocyte proliferation, being primarily useful in the field of immunoregulation and immunotherapy, are described. Specifically, Zot and zonulin inhibit antigen-presenting cell-mediated antigen-specific lymphocyte proliferation in a dose-dependent manner. This effect is associated with the presence of a macrophage surface receptor to which Zot binds in a specific and saturable way. This down-regulation of the immune response is, at least in part, associated with a decreased uptake of antigen.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L110 ANSWER 35 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:53692 HCAPLUS
DOCUMENT NUMBER: 132:103730
TITLE: Human FAST-1 gene and its use for screening **cancer** drugs as TGF-β modulators
INVENTOR(S): Zhou, Shibin; Zawel, Leigh; Vogelstein, Bert; Kinzler, Kenneth W.
PATENT ASSIGNEE(S): The Johns Hopkins University, USA
SOURCE: PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000002910	A2	20000120	WO 1999-US13764	19990618 <--
WO 2000002910	A3	20000316		

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ,

MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 6110738	A	20000829	US 1998-113309	19980710
AU 9945770	A1	20000201	AU 1999-45770	19990618 <--
US 6225441	B1	20010501	US 2000-521109	20000307 <--
PRIORITY APPLN. INFO.:			US 1998-113309 A1	19980710 <--
			WO 1999-US13764 W	19990618 <--

ED Entered STN: 23 Jan 2000
AB The invention isolated human forkhead activin signal transducer gene (hFAST-1) and includes tools for investigating the TGF-β signaling pathway and screening for **anticancer** compds. which modulate the action of TGF-β. Human FAST-1 was identified through the homol. search of Xenopus counterpart (xFAST-1) and mapped to chromosome 8q24. The hFAST-1 protein, containing forkhead DNA-binding domain, nuclear localization domain and Smad2 binding domain, binds to human Smad2 and activates an activin response element (ARE). The ARE motif is important for the activation of genes responsive to ligands of the TGF-β family and hFAST-1 activation of ARE is dependent on endogenous Smad4 and stimulation of the TGF-β receptor.

L110 ANSWER 36 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:811274 HCAPLUS
DOCUMENT NUMBER: 132:59187
TITLE: Anti-angiogenic proteins and methods of use thereof
INVENTOR(S): Kalluri, Raghuram
PATENT ASSIGNEE(S): Beth Israel Deaconess Medical Center, USA
SOURCE: PCT Int. Appl., 117 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9965940	A1	19991223	WO 1999-US13737	19990617 <--
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2331332	AA	19991223	CA 1999-2331332	19990617 <--
AU 9945755	A1	20000105	AU 1999-45755	19990617 <--
AU 753249	B2	20021010		
EP 1086129	A1	20010328	EP 1999-928762	19990617 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002517999	T2	20020625	JP 2000-554765	19990617 <--
PRIORITY APPLN. INFO.:			US 1998-89689P P	19980617 <--
			US 1999-126175P P	19990325 <--
			WO 1999-US13737 W	19990617 <--

ED Entered STN: 24 Dec 1999
AB Proteins with anti-angiogenic properties are disclosed, and methods of

using those proteins to inhibit angiogenesis are explained.
REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L110 ANSWER 37 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:388287 HCAPLUS
DOCUMENT NUMBER: 131:41277
TITLE: Mutants of endostatin, "em 1" having anti-angiogenic activity and methods of use thereof
INVENTOR(S): Sukhatme, Vikas P.
PATENT ASSIGNEE(S): Beth Israel Deaconess Medical Center, USA
SOURCE: PCT Int. Appl., 105 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE	
WO 9929855	A1	19990617	WO 1998-US26057		19981208	<--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, US, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM						
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG						
CA 2313251	AA	19990617	CA 1998-2313251		19981208	<--
AU 9917180	A1	19990628	AU 1999-17180		19981208	<--
AU 742866	B2	20020117				
EP 1037983	A1	20000927	EP 1998-962006		19981208	<--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI						
JP 2002503449	T2	20020205	JP 2000-524427		19981208	<--
PRIORITY APPLN. INFO.:			US 1997-67888P	P	19971208	<--
			US 1998-82663P	P	19980422	<--
			US 1998-108536P	P	19981116	<--
			WO 1998-US26057	W	19981208	<--

ED Entered STN: 23 Jun 1999
AB Described herein are novel mutants of endostatin, one of which, designated "EM 1", has anti-angiogenic activity similar or superior to that of wild type endostatin. The invention relates to the discovery of an isolated anti-angiogenic peptide, wherein the C-terminal end of the peptide comprises the amino acid sequence SYIVLCIE, which has anti-angiogenic properties. Designated "EM 1", this protein comprises a mutated endostatin protein, where the mutation comprises a deletion of nine consecutive amino acids from the C-terminus of the mutated endostatin protein (e.g., NSFMTSFSK). EM 1 terminates in the amino acid sequence SYIVLCIE. The invention also comprises isolated polynucleotides encoding EM 1, operably linked to expression sequence, and host cells transformed with such a construct. Antibodies to EM 1 are also disclosed. The invention also relates to processes for producing EM 1, fusion proteins containing EM 1, and compns. comprising EM 1 or fusion products thereof. The invention also discloses methods of producing polypeptides encoding EM 1.
REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L110 ANSWER 38 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:317194 HCAPLUS
DOCUMENT NUMBER: 130:343017
TITLE: Method of inhibiting side effects of pharmaceutical compositions containing amphiphilic vehicles or drug carrier molecules
INVENTOR(S): Szebeni, Janos; Alving, Carl R.
PATENT ASSIGNEE(S): Walter Reed Army Institute of Research, USA
SOURCE: PCT Int. Appl., 82 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 9922759	A1	19990514	WO 1998-US23280		19981030 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG					
AU 9912978	A1	19990524	AU 1999-12978		19981030 <--
EP 996461	A1	20000503	EP 1998-956455		19981030 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI					
US 2002136759	A1	20020926	US 1998-183375		19981030 <--
PRIORITY APPLN. INFO.:			US 1997-63876P	P	19971031 <--
			WO 1998-US23280	W	19981030 <--

ED Entered STN: 24 May 1999
AB Toxicity and other unwanted effects caused by (a) solvents or emulsifiers for pharmaceuticals which contain amphiphilic mols. such as polyethoxylated oils or (b) drug vehicles containing amphiphilic mols. such as phospholipids are inhibited or prevented by use of a complement inhibitor such as soluble complement receptor type 1. Thus, Cremophor EL (polyethoxylated castor oil) and phospholipid liposomes, used in taxol formulations, caused significant complement activation in human serum by both classical and alternative pathways; this effect was potentiated by EtOH in the formulations. Injection of large multilamellar phosphatidylcholine-phosphatidylglycerol-cholesterol liposomes or liposome-encapsulated Hb into **pigs** induced pulmonary hypertension and a large increase in plasma level of TXB2 (the stable metabolite of TXA2); these effects were inhibited by murine anti-porcine complement C5a antibody GS1 (1.6 mg/kg), recombinant soluble complement receptor type 1 (0.2 or 2 mg/kg), or the cyclooxygenase inhibitor, indomethacin (5 mg/kg).
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L110 ANSWER 39 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:184151 HCAPLUS
DOCUMENT NUMBER: 130:227705
TITLE: Oral vaccine comprising Lactobacillus species transformed with Helicobacter urease

INVENTOR(S): Tabaqchali, Soad; Wilks, Mark
PATENT ASSIGNEE(S): Queen Mary & Westfield College, UK
SOURCE: PCT Int. Appl., 23 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9911284	A1	19990311	WO 1998-GB2631	19980902 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9888779	A1	19990322	AU 1998-88779	19980902 <--
PRIORITY APPLN. INFO.:			GB 1997-18616	19970902 <--
			WO 1998-GB2631	19980902 <--

ED Entered STN: 22 Mar 1999
AB The present invention relates to a vaccine comprising a Lactobacillus species that contains a nucleotide sequence that encodes a urease peptide capable of initiating an anti-urease humoral and/or cellular immune response upon administration to a mammalian species. The vaccine may be used to treat gastrointestinal disorders, such as gastritis, that are the result of infection by Helicobacter strains, for which urease is the most prominent protein component.
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L110 ANSWER 40 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:675103 HCAPLUS
DOCUMENT NUMBER: 129:286410
TITLE: Hormone/lytic peptides and therapeutic use in controlling **cancer**, viral infection, and autoimmune diseases and in inducing sterility
INVENTOR(S): Enright, Frederick M.; Jaynes, Jesse M.; Hansel, William; Koonce, Kenneth L.; McCann, Samuel M.; Yu, Wen H.; Melrose, Patricia A.; Foil, Lane D.; Elzer, Philip H.
PATENT ASSIGNEE(S): Board of Supervisors of Louisiana State University and Agricultural and Mechanical College, USA
SOURCE: PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9842365	A1	19981001	WO 1998-US6114	19980327 <--
W: CA, JP, US, US, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 975354	A1	20000202	EP 1998-913218	19980327 <--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

JP 2000514836	T2	20001107	JP 1998-546026	19980327	<--
CA 2302392	AA	19990311	CA 1998-2302392	19980901	<--
WO 9911282	A1	19990311	WO 1998-US18117	19980901	<--

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 9892138	A1	19990322	AU 1998-92138	19980901	<--
JP 2001514231	T2	20010911	JP 2000-508384	19980901	<--
US 6635740	B1	20031021	US 1999-381879	19990924	<--
US 6680058	B1	20040120	US 2000-486143	20000222	<--
US 2004018967	A1	20040129	US 2003-617561	20030711	<--

PRIORITY APPLN. INFO.:	US 1997-41009P	P	19970327 <--
	US 1997-869153	A2	19970604 <--
	US 1997-57456P	P	19970903 <--
	US 1997-92112P	P	19970604 <--
	WO 1998-US6114	W	19980327 <--
	WO 1998-US18117	W	19980901 <--
	US 1999-381879	A1	19990924 <--

ED Entered STN: 26 Oct 1998

AB Amphipathic lytic peptides are ideally suited to use in a ligand/cytotoxin combination to specifically inhibit cells that are driven by or are dependent upon a specific ligand interaction; for example, to induce sterility or long-term contraception, or to attack **tumor** cells, or to selectively lyse virally-infected cells, or to attack lymphocytes responsible for autoimmune diseases. The peptides act directly on cell membranes, and need not be internalized. Administering a combination of gonadotropin-releasing hormone (GnRH) (or a GnRH agonist) and a membrane-active lytic peptide produces long-term contraception or sterilization in animals in vivo. Administering in vivo a combination of a ligand and a membrane-active lytic peptide kills cells with a receptor for the ligand. The compds. are relatively small, and are not antigenic. Lysis of gonadotropes has been observed to be very rapid (on the order of ten minutes). Lysis of **tumor** cells is rapid. The two components -the ligand and the lytic peptide- may optionally be administered as a fusion peptide, or they may be administered sep., with the ligand administered slightly before the lytic peptide, to activate cells with receptors for the ligand, and thereby make those cells susceptible to lysis by the lytic peptide. The compds. may be used in gene therapy to treat **malignant** or non-**malignant tumors**, and other diseases caused by clones or populations of "normal" host cells bearing specific receptors (such as lymphocytes), because genes encoding a lytic peptide or encoding a lytic peptide/peptide hormone fusion may readily be inserted into hematopoietic stem cells or myeloid precursor cells.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L110 ANSWER 41 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER:	1997:740426 HCAPLUS
DOCUMENT NUMBER:	128:53199
TITLE:	Cationic virosomes as transfer system for genetic material

INVENTOR(S): Walti, Ernst Rudolf; Gluck, Reinhard; Klein, Peter
PATENT ASSIGNEE(S): Nika Health Products Limited, Liechtenstein
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9741834	A1	19971113	WO 1997-EP2268	19970504 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2253561	AA	19971113	CA 1997-2253561	19970504 <--
AU 9727766	A1	19971126	AU 1997-27766	19970504 <--
AU 710170	B2	19990916		
EP 902682	A2	19990324	EP 1997-921852	19970504 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
CN 1225007	A	19990804	CN 1997-196232	19970504 <--
BR 9709224	A	19990810	BR 1997-9224	19970504 <--
NZ 332666	A	20000526	NZ 1997-332666	19970504 <--
JP 2000509404	T2	20000725	JP 1997-539526	19970504 <--
ZA 9703885	A	19981106	ZA 1997-3885	19970506 <--
HR 970234	B1	20020430	HR 1997-970234	19970507 <--
NO 9805137	A	19990104	NO 1998-5137	19981104 <--
KR 2000010780	A	20000225	KR 1998-708906	19981105 <--
US 6210708	B1	20010403	US 1999-414872	19991008 <--
NZ 504444	A	20001124	NZ 2000-504444	20000510 <--

PRIORITY APPLN. INFO.:	EP 1996-107282	A	19960508 <--
	NZ 1997-332666	A	19970504 <--
	WO 1997-EP2268	W	19970504 <--
	US 1998-171882	A2	19981230 <--

ED Entered STN: 24 Nov 1997
AB The present invention relates to a pos. charged virosome for efficient delivery of genetic material to resting or proliferating mammalian cells in vitro and in vivo. The virosome membrane contains cationic and/or polycationic lipids, at least one viral fusion peptide and preferably at least one cell-specific marker, advantageously selected from the group consisting of monoclonal antibodies, antibody fragments F(ab')2 and Fab', cytokines, and growth factors, for a selective detection and binding of target cells. The invention further relates to a method for the manufacture of the novel virosomes and to applications thereof, particularly for the manufacture of pharmaceutical compns. to treat **cancer** or **leukemia**.

L110 ANSWER 42 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:740418 HCAPLUS
DOCUMENT NUMBER: 128:43873
TITLE: Antiangiogenic peptides, polypeptides containing them, and methods for inhibiting angiogenesis
INVENTOR(S): Davidson, Donald J.; Wang, Jieyi; Gubbins, Earl J.

PATENT ASSIGNEE(S): Abbott Laboratories, USA
SOURCE: PCT Int. Appl., 78 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9741824	A2	19971113	WO 1997-US7700	19970505 <--
WO 9741824	A3	19980108		
W: AU, BR, CA, CN, CZ, HU, IL, JP, KR, MX, NZ				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5801146	A	19980901	US 1996-643219	19960503 <--
AU 9730606	A1	19971126	AU 1997-30606	19970505 <--
AU 724077	B2	20000914		
EP 910571	A2	19990428	EP 1997-925478	19970505 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, FI				
CN 1223690	A	19990721	CN 1997-195989	19970505 <--
BR 9708911	A	19990803	BR 1997-8911	19970505 <--
NZ 332319	A	20000929	NZ 1997-332319	19970505 <--
JP 2002502235	T2	20020122	JP 1997-540162	19970505 <--
PRIORITY APPLN. INFO.:			US 1996-643219 A	19960503 <--
			US 1997-832087 A	19970403 <--
			WO 1997-US7700 W	19970505 <--

ED Entered STN: 24 Nov 1997
AB Mammalian kringle 5 fragments and kringle 5 fusion proteins are disclosed as compds. for treating angiogenic diseases. Methods and compns. for inhibiting angiogenic diseases are also disclosed.

L110 ANSWER 43 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:655428 HCAPLUS
DOCUMENT NUMBER: 127:304777
TITLE: Mammalian **tumor** necrosis factor α convertase, recombinant expression and purification, and screening for hydroxamic acid derivative or other inhibitors useful for disease treatment
INVENTOR(S): McGeehan, Gerard M.; Becherer, James David; Moss, Marcia L.; Schoenen, Frank J.; Rocque, Warren J.; Chen, Wen-Ji; Didsbury, John R.; Jin, Shiow-Lian Catherine
PATENT ASSIGNEE(S): Glaxo Group Limited, UK; McGeehan, Gerard M.; Becherer, James David; Moss, Marcia L.; Schoenen, Frank J.; Rocque, Warren J.; Chen, Wen-Ji; Didsbury, John R.; Jin, Shiow-Lian Catherine
SOURCE: PCT Int. Appl., 132 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9735538	A2	19971002	WO 1997-EP1497	19970325 <--
WO 9735538	A3	19971120		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ,				

LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL,
PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ,
VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB,
GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN,
ML, MR, NE, SN, TD, TG

CA 2249985	AA	19971002	CA 1997-2249985		19970325 <--
AU 9722913	A1	19971017	AU 1997-22913		19970325 <--
EP 900272	A2	19990310	EP 1997-915426		19970325 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI					
JP 2000507943	T2	20000627	JP 1997-534033		19970325 <--
PRIORITY APPLN. INFO.:			US 1996-620663	A	19960326 <--
			WO 1997-EP1497	W	19970325 <--

ED Entered STN: 15 Oct 1997
AB The present invention relates to **tumor** necrosis factor α (TNFα), and more specifically to the enzyme TNFα-convertase (TNFα-con) that can proteolytically convert TNFα precursor to mature TNFα. The present invention provides DNA sequences encoding mammalian TNFα-con and functional equivalent thereof, recombinant expression vectors comprising said DNA sequences, host cell lines comprising said expression vectors, inhibitors of TNFα-con, inhibitors modified for use as ligands for affinity purification of TNFα-con, and methods for treating diseases or conditions resulting from abnormal levels of TNFα in a mammalian subject. The general invention is exemplified by preparation of a biotinylated inhibitor of TNFα-con.

L110 ANSWER 44 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:326866 HCAPLUS
DOCUMENT NUMBER: 126:308798
TITLE: Chimeric DNA-binding/DNA methyltransferase nucleic acid and polypeptide and their uses
INVENTOR(S): Bestor, Timothy H.
PATENT ASSIGNEE(S): Trustees of Columbia University in the City of New York, USA; Bestor, Timothy H.
SOURCE: PCT Int. Appl., 97 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 9711972	A1	19970403	WO 1996-US15576		19960927 <--
W: AU, CA, JP, MX, US, US					
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
AU 9673781	A1	19970417	AU 1996-73781		19960927 <--
US 2002188103	A1	20021212	US 1998-51013		19981009 <--
PRIORITY APPLN. INFO.:			US 1995-4445P	P	19950928 <--
			US 1996-594866	A2	19960131 <--
			WO 1996-US15576	W	19960927 <--

ED Entered STN: 23 May 1997
AB The present invention provides a chimeric protein which comprises a mutated DNA methyltransferase portion and a DNA binding protein portion that binds sufficiently close to a promoter sequence of a target gene (which promoter sequence contains a methylation site) to specifically methylate the site and inhibit activity of the promoter and thus inhibit

expression of the target gene. This invention also provides for a method for inhibiting the expression of a target gene which includes contacting a promoter of the target gene with the chimeric protein, so as to specifically methylate the promoter sequence of the target gene thus inhibiting expression of the target gene.

L110 ANSWER 45 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:189747 HCAPLUS
DOCUMENT NUMBER: 120:189747
TITLE: **Leukemia** inhibitory factor-binding protein
INVENTOR(S): Nicola, Nicos Antony; Layton, Meredith; Metcalf, Donald; Simpson, Richard J.
PATENT ASSIGNEE(S): Amrad Corp. Ltd., Australia
SOURCE: PCT Int. Appl., 70 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9401464	A1	19940120	WO 1993-AU325	19930701 <--
W: AT, AU, BB, BG, BR, BY, CA, CH, CZ, DE, DK, ES, FI, GB, HU, JP, KP, KR, KZ, LK, LU, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SK, UA, US, VN				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9344127	A1	19940131	AU 1993-44127	19930701 <--
AU 662433	B2	19950831		
EP 651769	A1	19950510	EP 1993-914550	19930701 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 07508733	T2	19950928	JP 1993-502757	19930701 <--
US 6387875	B1	20020514	US 1994-331650	19941110 <--
PRIORITY APPLN. INFO.:			AU 1992-3265 A	19920701 <--
			WO 1993-AU325 A	19930701 <--

ED Entered STN: 16 Apr 1994
AB A **leukemia** inhibitory factor (LIF)-binding protein (LBP) obtained from mammalian serum is capable of inhibiting the ability of LIF to induce differentiation of M1 myeloid **leukemic** cells in vitro in a dose-dependent manner. The LBP binds LIF from other mammals to a greater extent compared to its ability to inhibit LIF from the source organism and so can be used in the quantitation of LIF and in therapeutics (no data). The protein was observed in mouse serum where it blocked the binding of inactivating antibody to LIF and formed a stable 110 kDa complex with radioiodinated mouse LIF (20 kDa). The was purified by affinity chromatog. against immobilized LIF. The protein was very unstable and completely inactivated by reagents used in protein purification Mouse LBP was more effective at inhibiting the action of human LIF on M1 cells than it was at inhibiting the action of mouse LIF. The protein appears to be the cleavage product of a cellular LIF receptor α-chain and is glycosidated, going from .apprx.90 kDa to .apprx.65 kDa after treatment with N-glycanase.

L110 ANSWER 46 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:208570 HCAPLUS
DOCUMENT NUMBER: 120:208570
TITLE: Chimeric receptor polypeptides containing human H13 proteins and uses thereof

INVENTOR(S): Meruelo, Daniel; Yoshimoto, Takayuki
PATENT ASSIGNEE(S): New York University, USA
SOURCE: PCT Int. Appl., 148 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9325682	A2	19931223	WO 1993-US5569	19930611 <--
WO 9325682	A3	19940317		
W: AU, CA, JP, US				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9345322	A1	19940104	AU 1993-45322	19930611 <--
EP 644936	A1	19950329	EP 1993-915287	19930611 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 07509599	T2	19951026	JP 1993-501727	19930611 <--
PRIORITY APPLN. INFO.:			US 1992-899075 A2	19920611 <--
			WO 1993-US5569 A	19930611 <--

ED Entered STN: 30 Apr 1994
AB A chimeric viral receptor polypeptide comprising 2 receptor polypeptide fragments from different species, resp., is disclosed. The receptor polypeptides can be modified for binding the viral envelop protein (env). The chimeric receptor polypeptide can be used to provide target cells-specific binding sites for specific viruses, viral vectors, and delivery vectors of therapeutic and /or diagnostic agents. The cDNA for human H13 protein that is highly homologous to the murine ecotropic retroviral receptor (ERR) was cloned and used for preparation of a chimeric receptor polypeptide. Substitution, deletion, or addition of H13 extracellular domains 3 and 4 with corresponding ERR amino acids provided the infectivity of ecotropic murine **leukemic** retrovirus. H13 mutants with substitutions at 242-Pro→Tyr and 244-Val→Glu, or 240-Gly→Val and 242-Pro→Tyr were prepared and their effects demonstrated.

L110 ANSWER 47 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 1
ACCESSION NUMBER: 1986:399375 BIOSIS
DOCUMENT NUMBER: PREV198682084855; BA82:84855
TITLE: **EQUINE** SARCOID BCG **IMMUNOTHERAPY** COMPARED TO CRYOSURGERY IN A PROSPECTIVE RANDOMIZED CLINICAL TRIAL.
AUTHOR(S): KLEIN W R [Reprint author]; BRAS G E; MISDORP W; STEERENBERG P A; DE JONG W H; TIESJEMA R H; KERSJES A W; RUITENBERG E J
CORPORATE SOURCE: DEP OF GENERAL AND LARGE ANIMAL SURGERY, STATE UNIV UTRECHT, YALELAAN 1, 3584 CM UTRECHT, THE NETHERLANDS
SOURCE: Cancer Immunology Immunotherapy, (1986) Vol. 21, No. 2, pp. 133-140.
CODEN: CIIMDN. ISSN: 0340-7004.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 4 Oct 1986
Last Updated on STN: 4 Oct 1986
ED Entered STN: 4 Oct 1986
Last Updated on STN: 4 Oct 1986

AB A total of 30 **horses** with single or multiple sarcoid **tumors** of the skin were randomly divided into three treatment groups: (i) cryosurgical treatment, (ii) intralesional **immunotherapy** with a live BCG vaccine, (iii) intralesional **immunotherapy** with a BCG cell wall preparation. Complete **tumour** regression was obtained in all 10 crysurgically treated **horses**, in 6 of 10 live BCG treated **horses**, and in 7 of 10 BCG cell wall treated **horses**. One live BCG and 2 BCG cell wall treated **horses** showed partial **tumour** regression of more than 50% of the **tumour** area. Eleven **horses** with sarcoid **tumours** were not eligible for random allocation in the trial because unfavourable site or size of the **tumour** precluded cryosurgical treatment. These animals were treated with BCG cell wall vaccine except for 1 animal, which was treated with live BCG. In 4 cases this treatment was combined with cytoreductive surgery of the **tumour**. In this prognostically unfavourable group 8 animals showed complete **tumour** regression and 3 animals did not respond. Regression after BCG **immunotherapy** appeared to correlate with size (large **tumours** worse response) and localization of the sarcoid (less favourable results in the limb), and increase in peripheral blood leucocytes after the first injection. **Horses** with a positive delayed type hypersensitivity reaction to PPD before the start of treatment showed a tendency to more favourable prognosis than PPD negative **horses**. No correlation was present between regression and single or multiple presence of sarcoids, increase in body temperature after injection BCG and the formation of specific **antibodies** to BCG. None of the cured animals have shown **tumour** recurrence 3 to 40 months following treatment.

L110 ANSWER 48 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 2

ACCESSION NUMBER: 1978:173077 BIOSIS
DOCUMENT NUMBER: PREV197865060077; BA65:60077
TITLE: LYSIS OF **FELINE** LYMPHOMA CELLS BY COMPLEMENT DEPENDENT **ANTIBODIES** IN **FELINE** LEUKEMIA VIRUS CONTACT **CATS** CORRELATION OF LYSIS AND **ANTIBODIES** TO **FELINE** ONCORNAVIRUS ASSOCIATED CELL MEMBRANE ANTIGEN.
AUTHOR(S): GRANT C K [Reprint author]; ESSEX M; PEDERSEN N C; HARDY W D JR; STEPHENSON J R; COTTER S M; THEILEN G H
CORPORATE SOURCE: DEP MICROBIOL, HARV SCH PUBLIC HEALTH, 665 HUNTINGTON AVE, BOSTON, MASS 02115, USA
SOURCE: Journal of the National Cancer Institute, (1978) Vol. 60, No. 1, pp. 161-166.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB Sera were collected from normal **cats**, from **cats** exposed to feline leukemia virus (FeLV) infection and from sick animals including many with leukemias and lymphomas. In blind studies, the sera (346 samples) were tested for lytic **antibodies** with the use of **cat** complement in a 20 h 51Cr-release test, and for **antibodies** to feline oncornavirus associated cell membrane antigen (FOCMA) by the membrane immunofluorescence test. In both assays the target cells were FL74, a **cat** lymphoblastoid line which replicates FeLV. Correlation of presence or absence of **antibodies** detected by both tests was 91% overall and 100% for the 93 samples that contained lytic **antibody** predominantly at a titer of ≥ 1:25 and anti-FOCMA **antibody** at a titer of ≥ 1:16.

Complement-dependent and anti-FOCMA **antibodies** were detected in sera from viremic **cats**; by radioimmunoprecipitation these sera did not contain detectable **antibodies** to the major envelope or core proteins of FeLV (gp70 and p30), nor did they contain **antibodies** to the endogenous **cat** oncornavirus RD114. Immune sera that lysed FL74 target cells, which replicate A, B and C virus subgroups, also lysed F422 cells which replicate only a subgroup virus. Complement-dependent **antibodies** were detected in sera from laboratory-bred normal **cats** only after contact exposure to **cats** infected with FeLV; lytic **antibodies** appeared between 8-32 wk after exposure commenced. Appearance of lytic **antibody** coincided with 1st evidence in blood smears of virus infection, and prolonged high **antibody** titers were maintained, whether or not the leukemia virus infection persisted. The incidence of detection of complement-dependent **antibodies** was ≤ 2% in sera from **cats** maintained in controlled FeLV-free environments, 25% in sera from a randomized sampling of privately owned **cats**, 36-45% in sera from virus-infected **cats** of leukemia-cluster households and 8% in sera from **cats** with leukemia or lymphoma. Exposure of **cats** to horizontal FeLV infection induced **antibodies** that lysed **cat** lymphoma cells slowly with **cat** complement; these **antibodies** were similar to anti-FOCMA **antibodies** which have a proved **antitumor** and immune surveillance function in vivo.

L110 ANSWER 49 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 1999:339512 BIOSIS
DOCUMENT NUMBER: PREV199900339512
TITLE: GD3 ganglioside **antibody** augments **tumoricidal** capacity of **canine** blood mononuclear cells by induction of interleukin 12.
AUTHOR(S): Helfand, Stuart C. [Reprint author]; Dickerson, Erin B.; Munson, Keith L.; Padilla, Marcia L.
CORPORATE SOURCE: School of Veterinary Medicine, Department of Medical Sciences, University of Wisconsin-Madison, 2015 Linden Drive West, Madison, WI, 53706, USA
SOURCE: Cancer Research, (July 1, 1999) Vol. 59, No. 13, pp. 3119-3127. print.
CODEN: CNREA8. ISSN: 0008-5472.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Aug 1999
Last Updated on STN: 24 Aug 1999

ED Entered STN: 24 Aug 1999
Last Updated on STN: 24 Aug 1999

AB Monoclonal **antibody** R24 recognizes ganglioside GD3 expressed on the cell surfaces of some **tumor** cells and on a subset of human T lymphocytes. Binding of R24 to these lymphocytes induces proliferation, cytokine production, and activation of intracellular signaling pathways. In the current report, we investigated expression of gangliosides by canine mononuclear immune cells and studied the ability of antiganglioside **antibody** to activate these cells using **tumor** cell killing as a measure of activation. A subset of canine monocytes, but not lymphocytes, was found to express gangliosides GD3 and GD2 as determined by the binding of monoclonal **antibodies** R24 and 14.G2a, respectively. Only R24 augmented the **tumoricidal** potential of fresh canine peripheral blood mononuclear cells (PBMCs) against **tumor** cell lines that did not express surface gangliosides GD3 or

GD2. The augmenting effect of R24 on PBMC-mediated **tumor** cytotoxicity required cooperation between monocytes and lymphocytes because there was no enhancement of cytotoxicity mediated by R24 combined with either monocytes or lymphocytes individually. The enhancing effect of R24 on canine PBMC-mediated **tumor** cytotoxicity was blocked by anti-interleukin (IL)-12 neutralizing **antibody**, suggesting that R24 binding to monocytes triggered IL-12 release, contributing to the observed **tumor** killing effects. Reverse transcription-PCR confirmed that the binding of R24 to canine monocytes induced transcription of mRNA for canine IL-12. These data indicate that monocytes can be activated for **tumoricidal** responses through a membrane structure associated with ganglioside GD3 triggered by the binding of R24 and that the mechanism for enhanced cytotoxicity is due to the production and secretion of IL-12.

L110 ANSWER 50 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 2000:3321 BIOSIS
DOCUMENT NUMBER: PREV200000003321
TITLE: The immunotherapeutic potential of activated **canine** alveolar macrophages and **antitumor** monoclonal **antibodies** in metastatic **canine** melanoma.
AUTHOR(S): Soergel, Steve A.; MacEwen, E. Gregory; Vail, David M.; Potter, David M.; Sondel, Paul M.; Helfand, Stuart C. [Reprint author]
CORPORATE SOURCE: School of Veterinary Medicine, University of Wisconsin-Madison, 2015 Linden Dr. W., Madison, WI, 53706, USA
SOURCE: Journal of Immunotherapy, (Sept., 1999) Vol. 22, No. 5, pp. 443-453. print.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Dec 1999
Last Updated on STN: 31 Dec 2001

ED Entered STN: 23 Dec 1999
Last Updated on STN: 31 Dec 2001

AB A variety of immune cell activators can enhance the cytotoxic effects of monocytes/macrophages including interferon-gamma (IFN-gamma) and muramyl peptides, which are under investigation for **cancer** therapy in humans and **dogs**. Pulmonary alveolar macrophages (PAMs) in particular, are strategically located within the lung and provide a potential defense against **cancer** cells metastatic to the lung. For this reason, we examined the in vitro cytotoxic potential of fresh and IFN-gamma-activated PAMs from normal **dogs** targeted to canine **malignant** melanoma cells with antiganglioside monoclonal **antibodies** (mAbs). Antiganglioside mAbs 14.G2a (anti-GD2) and R24 (anti-GD3), both in clinical trials for human neuroectodermal **tumors** including melanoma, significantly enhanced the cytotoxicity of canine melanoma mediated by canine PAMs. Further, the cytotoxicity mediated by recombinant canine IFN-gamma-activated canine PAMs, in combination with anti-GD2 ganglioside mAb 14.G2a, enhanced melanoma cytotoxicity above tha t seen with mAb 14.G2a alone. This documentation of **antibody**-dependent cellular cytotoxicity mediated by activated PAMs suggests that activation and targeting of resident pulmonary immune cells be pursued as a means to control pulmonary metastases.

L110 ANSWER 51 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 1997:403788 BIOSIS
DOCUMENT NUMBER: PREV199799709991
TITLE: Immunologically specific activation of a cephalosporin derivative of mitomycin C by monoclonal **antibody** beta-**lactamase** conjugates.
AUTHOR(S): Vrudhula, Vivekananda M. [Reprint author]; Svensson, Hakan P.; Senter, Peter D.
CORPORATE SOURCE: Bristol-Myers Squibb Pharm. Res. Inst., 3005 First Ave., Seattle, WA 98121, USA
SOURCE: Journal of Medicinal Chemistry, (1997) Vol. 40, No. 17, pp. 2788-2792.
CODEN: JMCMAR. ISSN: 0022-2623.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Sep 1997
Last Updated on STN: 24 Sep 1997

ED Entered STN: 24 Sep 1997
Last Updated on STN: 24 Sep 1997

AB The syntheses of two cephalosporin derivatives 2 and 3 of mitomycin C (1) containing 7-phenylacetamido and 7-delta-carboxybutanamido side chains, respectively, are described. These compounds were prepared for evaluation as cephalosporin prodrugs capable of being activated by mAb-beta-lactamase conjugates. In vitro cytotoxicity assays performed on H2987 lung **adenocarcinoma** and done 62 melanoma cell lines indicated that compound 2 was comparable in cytotoxicity to the parent drug. In an effort to improve upon the cytotoxic differential of 2, an alternative prodrug 3 containing a polar carboxyl group in the side chain of the cephalosporin moiety was prepared. Compound 3 consistently behaved as a prodrug and was approximately 40- and 10-fold less toxic than 1 toward H2987 and done 62, respectively. Determination of kinetic constants for hydrolysis by beta-lactamase from Enterobacter cloacae P99 indicated k-**cat** values of 476 +- 170 and 248 +- 15.1 s-1 for 2 and 3, respectively. The k-**cat**/K-m ratios for 2 and 3 were found to be approximately 9.7 and 2.1 mu-M/s, respectively. Comparison of these k-**cat**/K-m values with those obtained for similar cephalosporin derivatives of other **antitumor** agents demonstrated that compounds with delta-carboxybutanamido side chains generally have slightly diminished efficiency of enzymatic hydrolysis compared to the corresponding 7-phenylacetamido analog. It was also demonstrated that the less toxic prodrug 3 was activated in an immunologically specific manner by L6-F(ab')-beta-lactamase and 96.5-F(ab')-beta-lactamase conjugates, selective for H2987 and clone 62 cells, respectively.

L110 ANSWER 52 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1997:157321 BIOSIS
DOCUMENT NUMBER: PREV199799456524
TITLE: Adoptive **immunotherapy** in **canine** chimeras.
AUTHOR(S): Kolb, H. J. [Reprint author]; Guenther, W.; Schumm, M.; Holler, E.; Wilmanns, W.; Thierfelder, S.
CORPORATE SOURCE: Med. Klinik III, Klinikum Grosshadern, Univ. Muenchen, Marchioninistr. 15, 81377 Muechen, Germany
SOURCE: Transplantation (Baltimore), (1997) Vol. 63, No. 3, pp. 430-436.
CODEN: TRPLAU. ISSN: 0041-1337.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Apr 1997

Last Updated on STN: 15 Apr 1997

ED Entered STN: 15 Apr 1997
Last Updated on STN: 15 Apr 1997

AB Chimerism and tolerance after bone marrow transplantation provide excellent conditions for adoptive **immunotherapy** with T cells of the marrow donor. We studied adoptive **immunotherapy** in **dog** leukocyte antigen-identical canine littermate chimeras. Mixed chimeras were produced by conditioning treatment with total body irradiation of a dose of 10 Gy, a uniformly lethal dose in **dogs**, and infusion of between 1 times 10-8 and 2 times 10-8/kg mononuclear marrow cells treated with absorbed antithymocyte globulin for inactivation of T cells. Donors were of opposite sex. Persistent mixed chimerism was induced in six of nine **dogs**, chimerism was complete in one **dog**, and only transient in two **dogs**. Tolerance to donor skin grafts was demonstrated in eight **dogs**, including a **dog** without cytogenetic evidence of chimerism. Lymphocytes of the marrow donor (between 3.2 times 10-8/kg and 4.1 times 10-8/kg) were transfused at various times after transplantation. Nontransfused **dogs** survived without graft-versus-host disease (GVHD), whereas **dogs** transfused on days 1 and 2 and **dogs** transfused on days 21 and 22 developed GVHD and died. In contrast, **dogs** transfused on days 61 and 62 or later survived without GVHD. Chimerism converted from mixed to complete in six of six transfused **dogs** and in one of eight nontransfused **dogs** (P lt 0.005). Donor lymphocyte transfusions 2 years and 4.5 years after transplantation induced split chimerism with lymphoid cells of donor origin and **myeloid cells** of host origin in one **dog** and complete chimerism in the other **dog**. Before lymphocyte collection, donors were immunized against tetanus toxin. Seven days after lymphocyte transfusion, recipients were given booster injections of tetanus toxoid and primary immunization against diphtheria toxin. In transfused animals, **antibody** titers against tetanus were demonstrated already before the booster injection. Transfused animals developed higher titers of **antibody** against tetanus and diphtheria toxin than nontransfused animals. Donor lymphocytes converted mixed chimerism into complete chimerism without producing GVHD, when the transfusion was delayed for 2 months or later after transplantation. Transfusion of donor lymphocytes transferred immune reactivity against tetanus toxin and improved reactivity against diphtheria toxin as a new antigen.

L110 ANSWER 53 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 1997:75108 BIOSIS
DOCUMENT NUMBER: PREV199799381811
TITLE: Isolation and characterization of the **canine** melanoma antigen recognized by the murine monoclonal **antibody** IBF9 and its distribution in cultured **canine** melanoma cell lines.
AUTHOR(S): Oliver, J. L. [Reprint author]; Wolfe, L. G.; Lopez, M. K. [Reprint author]; Church-Bird, A.; Toivio-Kinnucan, M.; Dietrich, M. A.
CORPORATE SOURCE: Dep. Veterinary Pathology, Sch. Veterinary Med., Louisiana State Univ., Baton Rouge, LA 70803, USA
SOURCE: American Journal of Veterinary Research, (1997) Vol. 58, No. 1, pp. 46-52.
CODEN: AJVRAH. ISSN: 0002-9645.
DOCUMENT TYPE: Article
LANGUAGE: English

ENTRY DATE: Entered STN: 26 Feb 1997
Last Updated on STN: 26 Feb 1997
ED Entered STN: 26 Feb 1997
Last Updated on STN: 26 Feb 1997
AB Objective: To characterize the canine melanoma antigen recognized by the murine monoclonal **antibody** IBF9 as to its cellular location, molecular size, protein and glycogen contents, and distribution in cell lines. Sample Population: 7 cultured canine melanoma cell lines. Procedure: Molecular characteristics of the antigen were determined by western blotting, enzymatic digestion studies, and tunicamycin inhibition studies. Distribution of the antigen in the cultured melanoma cell lines was determined by flow cytometry. Results: The antigen consists of 2 proteins with molecular mass of 89 and 85 kd. Tunicamycin and enzymatic digestion studies indicated that these proteins contained little glycosylation. Immunogold and immunofluorescence studies localized the antigen to the cell surface. Antigen expression was consistent within each cell line, with gt 90% of the cells positive for all cell lines except 1 (80%). Percentage of positive cells and relative intensity of immunostaining were constant throughout all phases of the cell cycle. Conclusions: The antigen identified by MAB IBF9 is a well-conserved and highly expressed cell surface protein present during all phases of the cell cycle in all **malignant** canine melanoma cell lines examined. Clinical Relevance: Because of consistency in expression, the antigen may have potential for use in **dogs** for melanoma immunodiagnostics and **immunotherapy**.

L110 ANSWER 54 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1995:385876 BIOSIS
DOCUMENT NUMBER: PREV199598400176
TITLE: Binding of monoclonal **antitumor** ganglioside **antibodies** to **canine** monocytes.
AUTHOR(S): Helfand, S. C.; Munson, K. L.
CORPORATE SOURCE: Sch. Vet. Med., Univ. Wis., Madison, WI, USA
SOURCE: 9TH INTERNATIONAL CONGRESS OF IMMUNOLOGY. (1995) pp. 890. The 9th International Congress of Immunology.
Publisher: 9th International Congress of Immunology, San Francisco, California, USA.
Meeting Info.: Meeting Sponsored by the American Association of Immunologists and the International Union of Immunological Societies. San Francisco, California, USA. July 23-29, 1995.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Sep 1995
Last Updated on STN: 1 Sep 1995
ED Entered STN: 1 Sep 1995
Last Updated on STN: 1 Sep 1995

L110 ANSWER 55 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1995:348901 BIOSIS
DOCUMENT NUMBER: PREV199598363201
TITLE: Therapeutic effects of diethylcarbamazine and 3'-azido-3'-deoxythymidine on **feline** leukemia virus lymphoma formation.
AUTHOR(S): Nelson, Phillip; Sellon, Rance; Novotney, Carol; Devera, Cristina; Davidian, Marie; English, Robert; Tompkins, Mary;

Tompkins, Wayne [Reprint author]
CORPORATE SOURCE: Dep. Microbiol. Pathol. Parasitol., Colege Vet. Med., North Carolina State University, 4700 Hillsborough St., Raleigh, NC 27606, USA
SOURCE: Veterinary Immunology and Immunopathology, (1995) Vol. 46, No. 1-2, pp. 181-194.
CODEN: VIIMDS. ISSN: 0165-2427.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Aug 1995
Last Updated on STN: 10 Aug 1995
ED Entered STN: 10 Aug 1995
Last Updated on STN: 10 Aug 1995
AB Twenty-four specific pathogen-free kittens were infected with the Rickard strain of feline leukemia virus (FeLV-R). The kittens were divided into four equal groups and were orally administered either a high dose of diethylcarbamazine (DECH, 12 mg kg-1), a low dose of diethylcarbamazine (DECL, 3 mg kg-1), 3'-azido-3'-deoxythymidine (AZT, 15 mg kg-1, b.i.d.), or a placebo (250 mg granular dextrose) daily for 10 weeks. Blood was collected at 2-week intervals for complete blood counts (CBC) and flow cytometric analysis (FACS) of peripheral blood lymphocytes (PBL). Plasma was assayed for **antibodies** to FeLV gp70 and for FeLV p27 antigen using ELISA assays. For FACS analysis, lymphocytes were incubated with monoclonal **antibodies** to feline Pan T, CD8+, CD4+, and B cell (Anti-Ig) antigens. In the placebo treated **cats**, FeLV-R infection caused an early (2 weeks p.i.) and persistent decrease in leukocyte numbers attributable primarily to a decrease in neutrophil numbers and a secondary lesser decrease in B and CD4+ lymphocyte numbers. The DEC-treated groups showed a delayed but similar leukopenia by 4 weeks p.i. The lymphopenia in the DEC groups (primarily B cells and CD4+ cells) was reversed by 10 weeks p.i. The lymphopenia in the DEC groups (primarily B cells and CD4+ cells) was reversed by 10 weeks p.i., but the neutropenia persisted. AZT treatment inhibited FeLV-R-induced lymphopenia but did not prevent a reduction in neutrophil numbers. A marked p27 antigenemia that peaked at 4 weeks p.i. was noted in the placebo treated **cats** and in most **cats** (11/12) treated with either dose of DEC. However, AZT significantly inhibited the p27 antigenemia and all **cats** were negative for p27 antigen between 6 and 10 weeks of treatment. In general, placebo treated **cats** as well as DECH and DECL **cats** had low levels of **antibody** to gp70 throughout the study, suggesting FeLV-R-induced immunosuppression. In contrast, significantly higher titers of anti-gp70 **antibodies** were seen in AZT-treated **cats** at 6 weeks p.i., and were maintained.

L110 ANSWER 56 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1994:548535 BIOSIS
DOCUMENT NUMBER: PREV199598008083
TITLE: Potential to involve multiple effector cells with human recombinant interleukin-2 and **antiganglioside** monoclonal **antibodies** in a **canine** **malignant** melanoma **immunotherapy** model.
AUTHOR(S): Helfand, Stuart C. [Reprint author]; Soergel, Steve A.; Donner, Robin L.; Gan, Jacek; Hank, Jacquelyn A.; Lindstrom, Mary J.; Sondel, Paul M.
CORPORATE SOURCE: Sch. Vet. Med., Univ. Wis.-Madison, 2015 Linden Dr. West, Madison, WI 53706, USA
SOURCE: Journal of Immunotherapy with Emphasis on Tumor Immunology,

(1994) Vol. 16, No. 3, pp. 188-197.
ISSN: 1067-5582.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 Dec 1994
Last Updated on STN: 22 Dec 1994
ED Entered STN: 22 Dec 1994
Last Updated on STN: 22 Dec 1994
AB Human **tumors** originating from neuroectodermal cells such as **malignant** melanoma and neuroblastoma express high levels of disialogangliosides GD2 and GD3, making these antigens ideal for targeting by monoclonal **antibodies** (Mabs). The purpose of this study was to investigate expression and targeting of gangliosides on canine melanoma. Using immunohistochemical methods, we analyzed the expression of disialogangliosides GD2 and GD3 on canine oral **malignant** melanomas with murine Mabs 14.G2a and R24 that recognize GD2 and GD3 disialogangliosides, respectively, on human **tumors**. We also assessed the ability of Mab 14.G2a (and its mouse-human chimera, ch 14.18) to mediate **antibody**-dependent cellular cytotoxicity (ADCC) in vitro against a canine **malignant** melanoma cell line with human recombinant interleukin-2 (IL-2) activated canine peripheral blood lymphocytes (PBL), or canine neutrophil effector cells. Our data show that Mabs 14.G2a and R24 recognized fresh frozen canine oral melanoma. Mabs 14.G2a or ch 14.18, or IL-2, potentiated lysis of the canine **malignant** melanoma cell line by canine PBL. The killing effect observed using the combination of either Mab with IL-2 was additive. Mab 14.G2a mediated potent ADCC of canine melanoma by canine neutrophils. These studies indicate that disialogangliosides are expressed on fresh canine melanoma cells. Mabs reactive with these antigens can target and trigger **tumor** killing by multiple canine effector populations and IL-2 can potentiate these effects by canine lymphocytes. Thus, canine oral **malignant** melanoma, a spontaneously occurring, metastatic **cancer** in the **dog**, may be a relevant animal model to investigate combination **immunotherapy** using **antitumor** Mab and IL-2.

L110 ANSWER 57 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1994:486022 BIOSIS
DOCUMENT NUMBER: PREV199497499022
TITLE: Spontaneous **canine** mammary **tumors**: A model for monoclonal **antibody** diagnosis and treatment of human breast **cancer**.
AUTHOR(S): Mottolese, Marcella; Morelli, Luisella; Agrimi, Umberto; Benevolo, Maria; Sciarretta, Francesco; Antonucci, Giovanni; Natali, Pier Giorgio [Reprint author]
CORPORATE SOURCE: Regina Elena Cancer Inst., Immunol. Lab., Via delle Messi D'Oro 156, 00158 Rome, Italy
SOURCE: Laboratory Investigation, (1994) Vol. 71, No. 2, pp. 182-187.
CODEN: LAINAW. ISSN: 0023-6837.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Nov 1994
Last Updated on STN: 9 Nov 1994
ED Entered STN: 9 Nov 1994
Last Updated on STN: 9 Nov 1994
AB Background: The increasing availability of monoclonal **antibodies** to human breast **tumor**-associated antigens offers a new means to

evaluate whether antigenic similarities exist between human and animal breast **tumors**. The study of the biology of spontaneous mammary **tumors** in the **dog**, one of the most frequent **neoplasias** in this domestic animal, may be of interest in providing an experimental model for the clinical management of human breast **cancer**. Experimental design: In order to assess whether antigenic similarities do occur between human and canine breast **cancer**, in the present study we have evaluated the immunohistochemical reactivity of two normal mammary glands and 62 benign and **malignant** canine breast **tumors** with a panel of six MoAbs, (HMFG-2, MBr1, B72.3, B6.2, X-10, B1.1) recognizing distinct human breast **tumor**-associated antigens. Results: The results of the present study indicate that mammary **neoplasias** in **dogs** display an antigenic phenotype comparable to that observed in female and male human breast lesions. As has already been demonstrated in humans, only three among the six monoclonal **antibodies** tested (B72.3, B6.2, and X-10) appear to discriminate benign from **malignant** canine mammary **tumors**. Conclusions: These findings demonstrate that (a) this veterinary **tumor** may represent a suitable model for imaging and **immunotherapy** studies of human breast **carcinoma** and (b) the triplet of reagents capable of distinguishing selectively transformed glandular epithelium may be useful in the immunocytochemical presurgical diagnosis of these canine **neoplasias**.

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ACCESSION NUMBER: 1993:390544 BIOSIS
DOCUMENT NUMBER: PREV199396065844
TITLE: Fractionated intravenous administration of yttrium-90-labeled B72.3 GYK-DTPA immunoconjugate in **beagle dogs**.
AUTHOR(S): Vriesendorp, Huibert M. [Reprint author]; Shao, Y.; Blum, Julia E.; Quadri, Syed M.; Williams, Jerry R.
CORPORATE SOURCE: Univ. Texas, M. D. Anderson Cancer Cent., Radiation Therapy, Box 97, 1515 Holcombe Blvd., Houston, TX 77030, USA
SOURCE: Nuclear Medicine and Biology, (1993) Vol. 20, No. 5, pp. 571-578.
ISSN: 0969-8051.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Aug 1993
Last Updated on STN: 3 Jan 1995

ED Entered STN: 23 Aug 1993
Last Updated on STN: 3 Jan 1995

AB B72.3, a monoclonal **antibody** with reactivity against human **adenocarcinomas**, was coupled with linker-chelator GYK-DTPA using carbohydrate mediated conjugation chemistry and radiolabeled with yttrium-90. Single and double intravenous injections of radioimmunoconjugate were compared for acute and late normal tissue toxicity in 15 beagle **dogs**. The second injection was given 4 or 8 days after the first. Pharmacokinetics of the radioimmunoconjugate in blood, blood marrow and urine were similar for first and second injections. Only bone marrow (acute) and liver (late) toxicity were observed. Both liver and bone marrow toxicity were decreased by fractionation of the injections. After double injections, the total equitoxic dose was 15 and 60% higher for bone marrow and liver toxicity, respectively. The mechanisms of normal tissue protection offered by

fractionated radioimmunoglobulin therapy (RIT) remain to be defined. Fractionated RIT will have a better therapeutic ratio than single injection RIT, if **antitumor** effects appear to be less susceptible to fractionation than normal tissues.

L110 ANSWER 59 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1993:390545 BIOSIS
DOCUMENT NUMBER: PREV199396065845
TITLE: Preclinical evaluation of intravenously administered indium-111- and yttrium-90-labeled B72.3 immunoconjugate (GYK-DTPA) in **beagle dogs**.
AUTHOR(S): Quadri, Syed M. [Reprint author]; Shao, Yi; Blum, Julia E.; Leichner, Peter K.; Williams, Jerry R.; Vriesendorp, Huibert M.
CORPORATE SOURCE: Univ. Texas, M. D. Anderson Cancer Cent., Exp. Radiotherapy-66, 1515 Holcombe Blvd., Houston, TX 77030, USA
SOURCE: Nuclear Medicine and Biology, (1993) Vol. 20, No. 5, pp. 559-570.
ISSN: 0969-8051.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Aug 1993
Last Updated on STN: 3 Jan 1995
ED Entered STN: 23 Aug 1993
Last Updated on STN: 3 Jan 1995
AB B72.3, a monoclonal **antibody** with reactivity against human **adenocarcinomas** was obtained from the Cytogen Corporation in the form of an immunoconjugate coupled with linker-chelator GYK-DTPA by using proprietary carbohydrate directed site specific chemistry. The immunoconjugate was radiolabeled with indium-111 or yttrium-90. A preclinical analysis was performed in 10 normal beagle **dogs**. The pharmacokinetics of intravenously administered indium- and yttrium-labeled immunoconjugates were compared serially in blood, bone marrow and urine samples. Compared to 90Y less of the 111In label ended up in urine and more was found in blood and bone marrow. Indium-labeled B72.3 GYK-DTPA had relatively higher uptake in most glandular tissues than 111In-labeled antiferritin immunoconjugate. Bone marrow toxicity was the dose limiting side effect after intravenous infusion of 90Y-labeled B72.3 GYK-DTPA. Toxicity was also observed in the liver but not in other organ systems. Recently other investigators obtained similar results with these immunoconjugates in human patients. A preclinical pharmacokinetic analysis of radioimmunoconjugates in beagle **dogs** provided useful information regarding bone marrow toxicity, liver toxicity and in vivo instability of the immunoconjugate. Data suggest that for future trials in human patients, a more stable chelated immunoconjugate for yttrium is needed to achieve less liver uptake and a better correlation with the 111In-labeled product than the 90Y-labeled B72.3 GYK-DTPA used in this investigation.

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ACCESSION NUMBER: 1993:525657 BIOSIS
DOCUMENT NUMBER: PREV199396139064
TITLE: **Antitumor** activities of a cephalosporin prodrug in combination with monoclonal **antibody**-beta-**lactamase** conjugates.
AUTHOR(S): Vrudhula, Vivekanada M.; Svensson, Hakan P.; Kennedy, Karen

A.; Senter, Peter D. [Reprint author]; Wallace, Philip M. [Reprint author]
CORPORATE SOURCE: Bristol-Myers Squibb Pharmaceutical Res. Inst., 3005 First Avenue, Seattle, WA 98121, USA
SOURCE: Bioconjugate Chemistry, (1993) Vol. 4, No. 5, pp. 334-340. CODEN: BCCHES. ISSN: 1043-1802.
DOCUMENT TYPE: Article
Errata
LANGUAGE: English
ENTRY DATE: Entered STN: 19 Nov 1993
Last Updated on STN: 13 Jan 1994
ED Entered STN: 19 Nov 1993
Last Updated on STN: 13 Jan 1994
AB 7-(Phenylacetamido)cephalosporin mustard (CM) and 7-(4-carboxybutanamido)cephalosporin mustard (CCM) were developed as **anticancer** prodrugs that could be activated site selectively by monoclonal **antibody**-beta-lactamase conjugates targeted to antigens present on **tumor** cell surfaces. Both CM and CCM were hydrolyzed by purified beta-lactamases from Escherichia coli (EC-beta-L), Bacillus cereus (BC-beta-L), and Enterobacter cloacae (ECl-beta-L). This resulted in the release of phenylenediamine mustard (PDM), a potent cytotoxic drug. The K-m and k-**cat** values of the reactions were determined, and it was found that ECl-beta-L effected the hydrolysis of CM and CCM more rapidly than the other enzymes. Conjugates of ECl-beta-L were prepared by reacting maleimide-substituted F(ab')-2 fragments of the monoclonal **antibodies** L6 and P1.17 to ECl-beta-L that had been modified with sulfhydryl groups. In vitro experiments indicated that CCM (IC-50 = 25-45 mu-M) was less toxic than PDM (IC-50 = 1.5 mu-M) to H2981 lung **adenocarcinoma** cells (L6 antigen positive, P1.17 antigen negative) and that immunologically specific prodrug activation took place when the cells were treated with L6-ECl-beta-L. In vivo experiments in nude mice demonstrated that CCM was less toxic than CM, and that both prodrugs were much less toxic than PDM. Neither CCM nor PDM exerted **antitumor** activity on subcutaneous H2981 **tumors** in vivo. However, a significant **antitumor** effect was obtained in mice that received L6-ECl-beta-L 96 h prior to the administration of CCM. The effect was immunologically specific (P lt 0.05), since a smaller degree of **antitumor** activity was obtained in mice that received the nonbinding control conjugate P1.17-ECl-beta-L prior to CCM. These studies demonstrate the potential therapeutic utility of monoclonal **antibody**-beta-lactamase conjugates for the activation of cephalosporin-containing prodrugs.

L110 ANSWER 61 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1991:116009 BIOSIS
DOCUMENT NUMBER: PREV199191063399; BA91:63399
TITLE: IN-VIVO DESTRUCTION OF **CANINE** LYMPHOMA MEDIATED BY MURINE MONOCLONAL **ANTIBODIES**.
AUTHOR(S): STEPLEWSKI Z [Reprint author]; ROSALES C; JEGLUM K A; MCDONALD-SMITH J
CORPORATE SOURCE: WISTAR INST, 36TH ST AT SPRUCE, PHILADELPHIA, PA 19104, USA
SOURCE: In Vivo (Attiki), (1990) Vol. 4, No. 4, pp. 231-234. CODEN: IVIVE4. ISSN: 0258-851X.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 27 Feb 1991
Last Updated on STN: 27 Feb 1991

ED Entered STN: 27 Feb 1991
Last Updated on STN: 27 Feb 1991
AB The effect of murine anti-canine lymphoma monoclonal **antibodies** (MAbs) on **tumor** cell lysis by thioglycolate activated murine macrophages in vitro and **tumor** growth inhibition in athymic mice was studied. All IgG1 and IgG2a MAbs tested were able to promote specific destruction of canine lymphoma 17-71 cell line by activated macrophages. A correlation between higher ADCC activity and MAb isotype was not clearly evident. In vivo IgG2a and IgG1 MAbs inhibited the growth of canine lymphoma. These results suggest that MAbs of IgG type have potential in **immunotherapy** of **dogs** with lymphoma since they have high **tumoricidal** activity in vivo.

L110 ANSWER 62 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1989:337702 BIOSIS
DOCUMENT NUMBER: PREV198988040702; BA88:40702
TITLE: **ANTIBODY**-RADIONUCLIDE CONJUGATES AS PART OF A **MYELOABLATIVE** PREPARATIVE REGIMEN FOR MARROW TRANSPLANTATION.
AUTHOR(S): APPELBAUM F R [Reprint author]; BROWN P; SANDMAIER B; BADGER C; SCHEUNING F; GRAHAM T; STORB R
CORPORATE SOURCE: ROOM 318, FRED HUTCHINSON CANCER RES CENT, DIV CLINICAL RES, 1124 COLUMBIA ST, SEATTLE, WASH 98104, USA
SOURCE: Blood, (1989) Vol. 73, No. 8, pp. 2201-2208.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 20 Jul 1989
Last Updated on STN: 27 Jul 1989
ED Entered STN: 20 Jul 1989
Last Updated on STN: 27 Jul 1989
AB The behaviors of an anti-Ia **antibody** (7.2) and an **antibody** directed at a lymphocyte adhesion molecule (S.5) radiolabeled with 131I were studied in normal **dogs**. **Antibody** 7.2 localized to spleen and, to a lesser extent, to marrow and lymph nodes. **Antibody** S.5 rapidly localized to marrow and spleen, achieving tissue/blood ratios > 6:1 within three hours of injection that were maintained for at least 48 hours. Prior treatment with cyclophosphamide (CY) markedly altered the distribution of S.5 but had much less effect on the distribution of 7.2 and almost no effect on the distribution of a control **antibody**. When animals were treated with increasing doses of 131I labeled to S.5, lethal myelosuppression occurred when a dose of 6 mCi/kg was reached. At this dose, the otherwise lethal effects of 131I could be reversed with autologous marrow transplant support.

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ACCESSION NUMBER: 1988:345049 BIOSIS
DOCUMENT NUMBER: PREV198835039891; BR35:39891
TITLE: **CANINE** LYMPHOMA A MODEL FOR CLINICAL TRIALS OF CYTOTOXIC MURINE MONOCLONAL **ANTIBODIES.**
AUTHOR(S): JEGLUM K A [Reprint author]; STEPLEWSKI Z
CORPORATE SOURCE: UNIV PENNSYLVANIA SCH VET MED, PHILADELPHIA, PA 19104, USA
SOURCE: Proceedings of the American Association for Cancer Research Annual Meeting, (1988) Vol. 29, pp. 421.
Meeting Info.: 79TH ANNUAL MEETING OF THE AMERICAN

ASSOCIATION FOR CANCER RESEARCH, NEW ORLEANS, LOUISIANA, USA, MAY 25-28, 1988. PROC AM ASSOC CANCER RES ANNU MEET. ISSN: 0197-016X.
DOCUMENT TYPE: Conference; (Meeting)
FILE SEGMENT: BR
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 26 Jul 1988
Last Updated on STN: 26 Jul 1988
ED Entered STN: 26 Jul 1988
Last Updated on STN: 26 Jul 1988

L110 ANSWER 64 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1988:505994 BIOSIS
DOCUMENT NUMBER: PREV198886126678; BA86:126678
TITLE: CYTOLYTIC ACTIVITY OF MURINE ANTI-**DOG** LYMPHOMA MONOCLONAL **ANTIBODIES** WITH **CANINE** EFFECTOR CELLS AND COMPLEMENT.
AUTHOR(S): ROSALES C [Reprint author]; JEGLUM K A; OBROCKA M; STEPLEWSKI Z
CORPORATE SOURCE: WISTAR INST, 36TH AT SPRUCE STREETS, PHILADELPHIA, PA 19104, USA
SOURCE: Cellular Immunology, (1988) Vol. 115, No. 2, pp. 420-428. CODEN: CLIMB8. ISSN: 0008-8749.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 22 Nov 1988
Last Updated on STN: 22 Nov 1988
ED Entered STN: 22 Nov 1988
Last Updated on STN: 22 Nov 1988
AB Peripheral blood leukocytes (PBL), nonadherent lymphocytes, and adherent monocytes separated from freshly isolated blood of 15 **dogs** were analyzed for their ability to mediate **antibody**-dependent cell-mediated cytotoxicity (ADCC) in combination with murine anti-**tumor** monoclonal **antibodies** (MAbs). Canine monocytes isolated from most donors by adherence to gelatin-fibronectin-coated plastic surface presented high ADCC activity against the canine lymphoma 17-71 **tumor** cell line in combination with antilymphoma MAbs 231 (IgG2a) and 234-2a (IgG2a) Canine lymphocytes generally showed lower ADCC activity than total PBL or monocytes. Canine PBL effector cells showed high ADCC activity against the human colorectal **carcinoma** SW948 cell line using the Y-6-specific MAb isotype switch variants 55-2 IgG3, 55-2 IgG1, 55-2 IgG2b, and 55-2 IgG2a. Analysis of the role of murine MAb isotypes on ADCC activity against **tumors** by canine cells using anti-human **tumor** class-switch variant MAbs and a panel of anti-canine lymphoma MAbs of different IgG subclass revealed the highest ADCC activity with MAbs of the IgG2a and IgG3 subclasses. IgG2a antilymphoma MAbs were also able to lyse **tumor** cells in complement-dependent cytotoxicity (CDC) assay. These results suggest the potential value of MAbs of IgG3 and IgG2a subclasses in **immunotherapy** against canine lymphoma.

L110 ANSWER 65 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1987:334817 BIOSIS
DOCUMENT NUMBER: PREV198784043760; BA84:43760
TITLE: 3' AZIDO-3'-DEOXYTHYMIDINE IN **FELINE** LEUKEMIA VIRUS INFECTED **CATS** A MODEL FOR THERAPY AND

PROPHYLAXIS OF AIDS.
AUTHOR(S): TAVARES L [Reprint author]; RONEKER C; JOHNSTON K; LEHRMAN S N; DE NORONHA F
CORPORATE SOURCE: DEP VETERINARY MICROBIOLOGY, NEW YORK STATE COLL VETERINARY MED, CORNELL UNIV, ITHACA, NEW YORK 14853, USA
SOURCE: Cancer Research, (1987) Vol. 47, No. 12, pp. 3190-3194.
CODEN: CNREA8. ISSN: 0008-5472.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 8 Aug 1987
Last Updated on STN: 8 Aug 1987
ED Entered STN: 8 Aug 1987
Last Updated on STN: 8 Aug 1987
AB Due to similarities between human immunodeficiency virus and feline leukemia virus, the etiological agents of acquired immunodeficiency syndromes in human and **cats**, the feline system was used as a model to conduct preliminary investigations as to the efficacy of the thymidine analogue 3'-azido-3'-deoxythymidine (AZT) as a therapeutic and preventive agent against retroviruses. In vitro evaluation of AZT cytotoxicity and its antiviral effects were conducted. Subsequently, 50 6-week-old specific pathogen free kittens were inoculated with a highly immunosuppressive strain of Rickard-Feline Leukemia Virus. These **cats** were randomly subdivided into smaller groups with initiation of AZT treatment at variable times postinfection. All animals were periodically monitored for circulating infectious virus particles and virus-neutralizing **antibodies**. Their clinical condition was closely followed throughout the 6 week AZT treatment phase and for several months thereafter. The results indicate that AZT prevents retrovirus infection if administered immediately following virus exposure, and may also reduce retrovirus replication if administered to previously infected animals. Some of the treated **cats** developed neutralizing **antibodies** against the virus and became resistant to subsequent viral challenge. Future trials with this drug, both for the prevention and treatment of retroviral diseases in humans and animals, are warranted.

L110 ANSWER 66 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1987:382090 BIOSIS
DOCUMENT NUMBER: PREV198784068587; BA84:68587
TITLE: **CANINE** LYMPHOMA-ASSOCIATED ANTIGENS DEFINED BY MURINE MONOCLONAL **ANTIBODIES**.
AUTHOR(S): STEPLEWSKI Z [Reprint author]; JEGLUM K A; ROSALES C; WEINTRAUB N
CORPORATE SOURCE: THE WISTAR, 36TH AND SPRUCE, PHILADELPHIA, PA 19104, USA
SOURCE: Cancer Immunology Immunotherapy, (1987) Vol. 24, No. 3, pp. 197-201.
CODEN: CIIMDN. ISSN: 0340-7004.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 5 Sep 1987
Last Updated on STN: 5 Sep 1987
ED Entered STN: 5 Sep 1987
Last Updated on STN: 5 Sep 1987
AB Lymphoma in **dogs** resembles human non-Hodgkin's lymphoma in pathological presentation, immunophenotype, and response to therapy, thus representing a good model for comparative studies with human disease. Monoclonal **antibodies** (MAbs) were derived from mice immunized

with a **dog** lymphoma cell line. Three MAbs were selected for further application in immunophenotyping and **immunotherapy**. The binding specificities, antigen characterization, and isotypes for these MAbs are described.

L110 ANSWER 67 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1982:202509 BIOSIS
DOCUMENT NUMBER: PREV198273062493; BA73:62493
TITLE: PROTECTION AGAINST **FELINE** LEUKEMIA BY VACCINATION WITH A SUBUNIT VACCINE.
AUTHOR(S): LEWIS M G [Reprint author]; MATHES L E; OLSEN R G
CORPORATE SOURCE: DEP VETERINARY PATHOL, COLL BIOLOGICAL SCI, OHIO STATE UNIV, COLUMBUS, OHIO 43210, USA
SOURCE: Infection and Immunity, (1981) Vol. 34, No. 3, pp. 888-894. CODEN: INFIBR. ISSN: 0019-9567.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
AB An effective vaccine against feline leukemia virus infection was developed by the collection and concentration of tissue culture medium harvested from a [feline] **tumor** cell line. Lymphoid cells were grown to near saturation density in a normal growth medium and then transferred to a serum-free medium. The serum-free medium was collected, concentrated and evaluated for its vaccine potential. **Cats** receiving the vaccine emulsified in complete Freund adjuvant developed high antiviral and **antitumor** titers and were protected (81%) againt virus challenge. **Cats** receiving the vaccine without an adjuvant developed lower **antibody** levels and lower protection (53%) from viremia. Age-matched and litter-matched controls developed no **antibody** to test antigens before the challenge, and 100% became persistently viremic after the challenge. Vaccination with the soluble **tumor** cell antigen vaccine proved successful in preventing the induction of feline leukemia virus infection.

L110 ANSWER 68 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1979:188177 BIOSIS
DOCUMENT NUMBER: PREV197967068177; BA67:68177
TITLE: COMPLEMENT AND **TUMOR ANTIBODY** LEVELS IN **CATS** AND CHANGES ASSOCIATED WITH NATURAL **FELINE** LEUKEMIA VIRUS INFECTION AND **MALIGNANT** DISEASE.
AUTHOR(S): GRANT C K [Reprint author]; PICKARD D K; RAMAIKA C; MADEWELL B R; ESSEX M
CORPORATE SOURCE: DEP MICROBIOL, HARV SCH PUBLIC HEALTH, BOSTON, MASS 02115, USA
SOURCE: Cancer Research, (1979) Vol. 39, No. 1, pp. 75-81. CODEN: CNREA8. ISSN: 0008-5472.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
AB Complement[C]-dependent **antibody** (CDA) and C levels were measured in sera from 314 **cats**. Many of the **cats** (65%) received prior natural exposure to horizontally transmitted feline leukemia virus (FeLV), and some of the **cats** (13%) had lymphoid **tumors**. In CDA assays, sera were tested against 51Cr-labeled **cat** lymphoma cells, and normal **cat** serum was used as a C source. In assays for total lytic C activity, the lymphoma cells were

presensitized with alloantiserum. CDA was frequently detected in sera from persistently viremic **cats**, suggesting no direct CDA-FeLV interaction, and lack of such **antibody**-virus interaction in sera was confirmed by contingency table analysis. CDA was infrequently detected in **cats** with **tumors**, and data analysis provided strong support for CDA-**tumor** interaction (P = 0.002), thereby supporting the concept that CDA has specific **antitumor** activity. Most individual **cat** C values were distributed normally, but a reduction in mean activity was found in FeLV-infected **cats** with detectable CDA. Wide variations in C activity occurred from week to week only in FeLV-infected animals, and severely depleted C levels were sometimes associated with FeLV-related anemia or lymphoid **cancers**. The possibility is discussed that some lymphoid **tumors** of **cats** develop and progress in the face of a CDA-mediated (anti-feline oncornavirus-associated cell membrane antigen) **tumor** immune response at times when C levels are depleted.

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ACCESSION NUMBER: 1979:162719 BIOSIS
DOCUMENT NUMBER: PREV197967042719; BA67:42719
TITLE: REGRESSION OF A **THYMUS** DERIVED CELL LYMPHOMA AFTER ADMINISTRATION OF ANTI THYMOCYTE GLOBULIN.
AUTHOR(S): FISHER R I [Reprint author]; KUBOTA T T; MANDELL G L; BRODER S; YOUNG R C
CORPORATE SOURCE: NATL CANCER INST, ROOM 12N226, BUILD 10, BETHESDA, MD 20014; USA
SOURCE: Annals of Internal Medicine, (1978) Vol. 88, No. 6, pp. 799-800.
CODEN: AIMEAS. ISSN: 0003-4819.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
AB A patient with Sezary syndrome developed a diffuse undifferentiated lymphoma of T [thymus-derived] cell origin. After becoming resistant to multiple chemotherapeutic agents [bleomycin, adriamycin, cyclophosphamide, vincristine, prednisone, 6-mercaptopurine, methotrexate and epipodophyllotoxin VP 16-213], the patient was treated with [**horse**] antithymocyte globulin. A 75% reduction in adenopathy and complete resolution of skin erythema was observed during an 8 day period. In addition the percent of circulating T cells and the ability of those cells to respond to phytohemagglutinin and concanavalin A were reduced after antithymocyte globulin therapy. The patient died of an intracerebral hemorrhage secondary to profound thrombocytopenia. **Tumor** lysis may be achieved by passive **antibody** therapy in certain advanced lymphomas.

L110 ANSWER 70 OF 84 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1978:153593 BIOSIS
DOCUMENT NUMBER: PREV197865040593; BA65:40593
TITLE: DI METHYL MYLERAN AND AUTOLOGOUS MARROW GRAFTING FOR THE TREATMENT OF SPONTANEOUS **CANINE** LYMPHOMA.
AUTHOR(S): WEIDEN P L [Reprint author]; STORB R; SHULMAN H; GRAHAM T C
CORPORATE SOURCE: DIV ONCOL, DEP MED, UNIV WASH SCH MED, SEATTLE, WASH 98195, USA
SOURCE: European Journal of Cancer, (1977) Vol. 13, No. 12, pp. 1411-1416.
CODEN: EJCAAH. ISSN: 0014-2964.

DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
AB Dimethyl myleran (DMM), an **antitumor** drug effective against several rodent and human **tumors**, has severe hematopoietic toxicity, but only moderate immunosuppressive activity in **dogs**. In the present study, 18 **dogs** with spontaneous lymphoma were treated with 7.5-10 mg/kg DMM followed by infusion of previously aspirated autologous marrow to protect against otherwise lethal hematopoietic toxicity. Eleven **dogs** died in less than 14 days, several of unexpectedly severe gastrointestinal toxicity. Seven **dogs** survived 15-95 (median 38) days. Only 1 **dog** achieved a transient complete clinical remission; 12 additional **dogs** showed a greater than 50% decrease in palpable adenopathy. All but 1 **dog** had histologic evidence of lymphoma at autopsy. Immune responses were studied in 6 **dogs** surviving greater than 30 days. **Antibody** formation to **sheep** red blood cells was delayed, but ultimately titers were similar to those of normal **dogs**. Production of lymphocytotoxic **antibody** was slightly enhanced while production of **antibody** to bacteriophage was slightly impaired. Although high dose DMM alone does not appear to be effective in achieving complete remission in **dogs** with lymphoma, the relative lack of immunosuppression following DMM preserves the potential usefulness of this drug in the design of chemoimmunotherapy protocols.

L110 ANSWER 71 OF 84 EMBASE on STN
ACCESSION NUMBER: 1998196003 EMBASE
TITLE: In vivo **tumor** transfection with **superantigen** plus **cytokine** genes induces **tumor** regression and prolongs survival in **dogs** with **malignant** melanoma.
AUTHOR: Dow S.W.; Elmslie R.E.; Willson A.P.; Roche L.; Gorman C.; Potter T.A.
CORPORATE SOURCE: Dr. T.A. Potter, Division of Basic Immunology, Natl. Jewish Medical and Res. Center, 1400 Jackson Street, Denver, CO 80206, United States
SOURCE: Journal of Clinical Investigation, (1 Jun 1998) 101/11 (2406-2414).
Refs: 44
ISSN: 0021-9738 CODEN: JCINAO
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English
AB In vivo transfection of established **tumors** with immunostimulatory genes can elicit **antitumor** immunity. Therefore, we evaluated the safety and efficacy of intratumoral injections of a bacterial **superantigen** with a **cytokine** gene in **dogs** with **malignant** melanoma, a spontaneous and highly **malignant canine tumor**. 26 **dogs** with melanoma were treated with lipid- complexed plasmid DNA encoding staphylococcal enterotoxin B and either GM- CSF or IL-2. **Dogs** were evaluated for treatment-associated toxicity, **tumor** responses, immunologic responses, and survival times. The overall response rate (complete or partial remissions) for all 26 **dogs** was 46% (12 of 26), and was highest in patients with smaller **tumors**.

Toxicity was minimal or absent in all **dogs**. Injected **tumors** developed marked infiltrates of CD4+ and CD8+ T cells and macrophages, and rumor regression was associated with development of high levels of **antitumor** cytotoxic T lymphocyte activity in peripheral blood lymphocytes. Survival times for animals with stage III melanomas treated by intratumoral gene therapy were prolonged significantly compared with animals treated with surgical **tumor** excision only. Thus, local **tumor** transfection with **superantigen** and **cytokine** genes was capable of inducing both local and systemic **antitumor** immunity in an outbred animal with a spontaneously developing **malignant tumor**.

L110 ANSWER 72 OF 84 EMBASE on STN

ACCESSION NUMBER: 1998376622 EMBASE
TITLE: **Tumour** therapy with **radiolabelled antibodies**: Optimisation of therapy.
AUTHOR: Vriesendorp H.M.; Quadri S.M.; Borchardt P.E.
CORPORATE SOURCE: Dr. H.M. Vriesendorp, Arlington Cancer Center, 906 W. Randol Mill Road, Arlington, TX 76012, United States. acchmv@ix.netcom.com
SOURCE: BioDrugs, (1998) 10/4 (275-293).
Refs: 86
ISSN: 1173-8804 CODEN: BIDRF4
COUNTRY: New Zealand
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The promise of radiolabelled immunoglobulin therapy (RIT) for selective patient friendly, **cancer** treatment has not yet been fulfilled. Only patients with haematological **malignancies** show sizable response rates after RIT. With solid **tumours**, intravenous administration of radiolabelled **antibodies** does not provide sufficient turnout targeting. However, intracompartmental administration may solve this problem, particularly if **tumour** reactive IgM is used. Clinical progress in RIT depends on understanding the important RIT variables: **antigen**, **antibody**, radioisotope, conjugation chemistry, activity escalation, fractionation and protein dose. These are reviewed and a new translational decision tree/flow diagram is presented that can limit analysis to the most important RIT variables for a particular disease. These variables may differ depending on the type and stage of **cancer**, but the guiding principles in RIT development remain the same: selectivity and accountability. The proper application of these principles leads to the definition of a new series of phase I, II, III studies. These studies are more appropriate for the clinical exploration of RIT and place an emphasis on therapeutic ratio rather than toxicity.

L110 ANSWER 73 OF 84 EMBASE on STN

ACCESSION NUMBER: 97189477 EMBASE
DOCUMENT NUMBER: 1997189477
TITLE: Toward **antibody**-directed enzyme prodrug therapy with the **T268G** mutant of human carboxypeptidase A1 and novel in vivo stable prodrugs of methotrexate.
AUTHOR: Smith G.K.; Banks S.; Blumenkopf T.A.; Cory M.; Humphreys

J.; Laethem R.M.; Miller J.; Moxham C.P.; Mullin R.; Ray P.H.; Walton L.M.; Wolfe III L.A.
CORPORATE SOURCE: G.K. Smith, Dept. of Molecular Biochemistry, Glaxo Wellcome Res. and Development, 5 Moore Dr., Research Triangle Park, NC 27709, United States. garysmith@glaxo.com
SOURCE: Journal of Biological Chemistry, (1997) 272/25 (15804-15816).
Refs: 49
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB **Antibody**-directed enzyme prodrug therapy (ADEPT) has the potential of greatly enhancing **antitumor** selectivity of **cancer** therapy by synthesizing chemotherapeutic agents selectively at **tumor** sites. This therapy is based upon targeting a prodrug-activating enzyme to a **tumor** by attaching the enzyme to a **tumor**-selective **antibody** and dosing the enzyme-**antibody** conjugate systemically. After the enzyme-**antibody** conjugate is localized to the **tumor**, the prodrug is then also dosed systemically, and the previously targeted enzyme converts it to the active drug selectively at the **tumor**. Unfortunately, most enzymes capable of this specific, **tumor** site generation of drugs are foreign to the human body and as such are expected to raise an immune response when injected, which will limit their repeated administration. We reasoned that with the power of crystallography, molecular modeling and site-directed mutagenesis, this problem could be addressed through the development of a human enzyme that is capable of catalyzing a reaction that is otherwise not carried out in the human body. This would then allow use of prodrugs that are otherwise stable in vivo but that are substrates for a **tumor**-targeted mutant human enzyme. We report here the first test of this concept using the human enzyme carboxypeptidase A1 (hCPA1) and prodrugs of methotrexate (MTX). Based upon a computer model of the human enzyme built from the well known crystal structure of bovine carboxypeptidase A, we have designed and synthesized novel bulky phenylalanine- and tyrosine- based prodrugs of MTX that are metabolically stable in vivo and are not substrates for wild type human carboxypeptidases A. Two of these analogs are MTX-α-3-cyclobutylphenylalanine and MTX-α-3-cyclopentyltyrosine. Also based upon the computer model, we have designed and produced a mutant of human carboxypeptidase A1, changed at position 268 from the wild type threonine to a glycine (hCPA1-T268G). This novel enzyme is capable of using the in vivo stable prodrugs, which are not substrates for the wild type hCPA1, as efficiently as the wild type hCPA1 uses its best substrates (i.e. MTX-α- phenylalanine). Thus, the κ(**cat**)/K(m) value for the wild type hCPA1 with MTX-α-phenylalanine is 0.44 .apprx.μM-1 S-1, and κ(**cat**)/K(m) values for hCPA1-T268G with MTX-α-3-cyclobutylphenylalanine and MTX. α-3-cyclopentyltyrosine are 1.8 and 0.16 μM-1 s-1, respectively. The cytotoxic efficiency of hCPA1-268G was tested in an in vitro ADEPT model. For this experiment, hCPA1-T268G was chemically conjugated to ING-1, an **antibody** that binds to the **tumor antigen** Ep-Cam, or to Campath-1H, an **antibody** that binds to the T and B cell **antigen** CDw52. These conjugates were then incubated with HT-29 human colon adenocarcinoma cells (which express Ep-Cam but not the Campath 1H **antigen**) followed by incubation of the cells with the

in vivo stable prodrugs. The results showed that the targeted ING-1:hCPA1-T268G conjugate produced excellent activation of the MTX prodrugs to kill HT-29 cells as efficiently as MTX itself. By contrast, the enzyme-Cam-path 1H conjugate was without effect. These data strongly support the feasibility of ADEPT using a mutated human enzyme with a single amino acid change.

L110 ANSWER 74 OF 84 EMBASE
on STN

ACCESSION NUMBER: 1998004765 EMBASE
TITLE: Preclinical analysis of **radiolabeled** anti-GD2 immunoglobulin G.
AUTHOR: Vriesendorp F.J.; Quadri S.M.; Flynn R.E.; Malone M.R.; Cromeens D.M.; Stephens L.C.; Vriesendorp H.M.
CORPORATE SOURCE: Dr. F.J. Vriesendorp, Department of Neurology, Univ. of Texas Health Science Center, 6431 Fannin, Houston, TX 77030, United States
SOURCE: Cancer, (1997) 80/12 SUPPL. (2642-2649).
Refs: 27
ISSN: 0008-543X CODEN: CANCAR
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 016 Cancer
023 Nuclear Medicine
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

AB BACKGROUND. Unlabeled murine monoclonal anti-GD2 immunoglobulin (Ig)G (14G2a) reactive with nervous system diganglioside and neuroblastoma, melanoma, and small cell lung **carcinoma** produces **tumor** regression. However, serious acute abdominal pain, paresthesia, hypotension and hypertension, syndrome of inappropriate secretion of antidiuretic hormone (SIADH), and occasional motor weakness occur. Studies in preclinical animal models can elucidate the mechanism of the observed neurotoxicity and lead to anti-GD2 **antibody** treatment with a higher therapeutic ratio. METHODS. One mg of 14G2a or control IgG was labeled with 1-2 mCi of indium-111 and administered intravenously to beagles (n = 8). In 2 **dogs**, additional high dose (200 mg) unlabeled 14G2a was given over 5 days. Whole body gamma camera images and SPECT scans were obtained repeatedly over 7 days. On Day 7, sciatic nerve conduction studies were performed, and after euthanasia radioactivity was determined in major organs. RESULTS. Unlabeled high dose 14G2a administered to mice, rats, or rabbits did not cause neurotoxicity within 3 weeks. GD2 **antigens** were shown by immunochemistry to be present in brain and peripheral nerve tissues of rodents and beagles. After in vivo administration of radiolabeled 14G2a, **canine** lymph nodes showed specific uptake, but only minimal radioactivity was found in the nervous system. **Dogs** that received additional high dose unlabeled 14G2a showed much higher lymph node uptake and follicular lymph node hyperplasia. Low motor response amplitudes on nerve conduction studies were noted. CONCLUSIONS. A radioisotope label on IgG and its visualization in a large series of animal models indicate that a low protein dose of anti-GD2 IgG will not cause neurologic side effects in patients. High protein dose anti-GD2 IgG may enhance **antineoplastic** effects and contribute to neurotoxicity through stimulation of normal lymphocytes with subsequent release of **cytokines**.

L110 ANSWER 75 OF 84 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 97000877 EMBASE
DOCUMENT NUMBER: 1997000877
TITLE: Proposal for translational analysis and development of clinical **radiolabeled** immunoglobulin therapy.
AUTHOR: Vriesendorp H.M.; Quadri S.M.; Jaeckle K.A.; Freedman R.S.; Cromeens D.M.
CORPORATE SOURCE: H.M. Vriesendorp, Dept. of Radiotherapy-Box 97, University of Texas, M.D. Anderson Cancer Center, 1515 Holcombe Boulevard, Houston, TX 77030, United States
SOURCE: Radiotherapy and Oncology, (1996) 41/2 (151-161). ISSN: 0167-8140 CODEN: RAONDT
PUBLISHER IDENT.: S 0167-8140(96)01829-4
COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 014 Radiology
016 Cancer
023 Nuclear Medicine
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Background and purpose: Radiolabeled immunoglobulin therapy (RIT) can be a selective, effective, low-toxicity outpatient **cancer** therapy. A consensus on the best approach for the preclinical and clinical development of RIT reagents needs to be developed. We report the M.D. Anderson **Cancer** Center prior experience in translating RIT from laboratory to clinic for the treatment of Hodgkin's disease and propose a flow diagram for the development of RIT for other **malignancies**. Material and methods: Three different animal models are described: nude mice bearing human **tumor** xenografts, normal beagle **dogs**, and normal rhesus monkeys. We produced and purified **antibodies** and prepared chelate-**immunoconjugates** reactive with six different human **tumor**-associated **antigens**. The Igs used were derived from rabbits, mice, and humans (human-derived RIT reagents being less immunogenic in human patients). Eighty patients with refractory Hodgkin's disease were treated with radiolabeled antiferritin. Results: We recommend a two-injection scheme using, (1) an indium-111-labeled radioimmunoconjugate for diagnosis, pharmacokinetic studies, and dosimetry, and (2) a yttrium-90-labeled radioimmunoconjugate for therapy. The animal models provide useful data on **tumor** targeting, radiotoxicology, and undesirable biodistributions. A 70% response rate is obtained in patients with advanced recurrent Hodgkin's disease. More extensive preclinical testing allows for safer and more effective clinical RIT studies. Conclusions: We recommend, (1) preclinical optimization of chelation chemistry, Ig size, Ig origin, route of administration, and fractionation, (2) new clinical Phase I-III studies more appropriate for RIT development than the classical Phase I-III studies used for the development of chemotherapeutic agents, and (3) more extensive preclinical testing of RIT reagents.

L110 ANSWER 76 OF 84 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 95060868 EMBASE
DOCUMENT NUMBER: 1995060868
TITLE: Generation of therapeutic T-lymphocytes after in vivo **tumor** transfection with an **allogeneic** class I major histocompatibility complex gene.

AUTHOR: Wahl W.L.; Strome S.E.; Nabel G.J.; Plautz G.E.; Cameron M.J.; San H.; Fox B.A.; Shu S.; Chang A.E.
CORPORATE SOURCE: Division of Surgical Oncology, Taubman Center, 1500 E. Medical Center Drive,Ann Arbor, MI 48109-0331, United States
SOURCE: Journal of Immunotherapy, (1995) 17/1 (1-11). ISSN: 1053-8550 CODEN: JOIME7
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
022 Human Genetics
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

AB In an effort to enhance the generation of **tumor**-reactive T-lymphocytes for adoptive immunotherapy, we examined the effects of in vivo transfection of an allogeneic major histocompatibility complex (MHC) class I gene (H-2K(s)) of the poorly immunogenic B16BL6 (BL6) melanoma of H-2b origin. Cells from lymph nodes (LNs) draining these **tumors** after transfection were assessed in adoptive immunotherapy experiments for **tumor** reactivity after sequential activation with anti-CD3 monoclonal **antibody** (mAb) followed by culture in interleukin (IL)-2. H-2K(s) lipofection of progressively growing BL6 subcutaneous **tumors** did not reduce **tumorigenicity**. However, in vivo lipofection of BL6 by intratumor inoculation or admixture of H-2K(s) cDNA/liposome complexes and **tumor** cells prior to inoculation resulted in enhanced development of sensitized T-lymphocytes in the draining LN, which mediated the reduction of the numbers of established 3-day parental lung metastases in six of six experiments. In subsequent studies, in vivo transfection of BL6 with naked H-2K(s) cDNA was found to be more effective than lipofection in eliciting sensitized T-cells in the draining LN. Admixture of liposomes alone or control plasmid DNA did not have an adjuvant effect similar to H-2K(s) cDNA. Relative **tumor** transfection efficiency was assessed by an indirect assay with the chloramphenicol acetyltransferase (**CAT**) reporter gene. BL6 **tumors** were more efficiently transfected by intratumor inoculation with naked cDNA compared with lipofection. In summary, in vivo allogenization of the poorly immunogenic BL6 **tumor** resulted in enhanced generation of therapeutic T-cells effective in the treatment of parental **tumor**.

L110 ANSWER 77 OF 84 EMBASE on STN
ACCESSION NUMBER: 86085795 EMBASE
DOCUMENT NUMBER: 1986085795
TITLE: **Myasthenia** gravis and polymyositis in a **dog** following fetal hematopoietic cell transplantation.
AUTHOR: Cain G.R.; Cardinet III G.H.; Cuddon P.A.; et al.
CORPORATE SOURCE: Laboratory for Energy-Related Health Research, University of California, Davis, CA 95616, United States
SOURCE: Transplantation, (1986) 41/1 (21-25). CODEN: TRPLAU
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 026 Immunology, Serology and Transplantation
025 Hematology
008 Neurology and Neurosurgery

LANGUAGE: English
AB Myasthenia gravis and focal polymyositis occurred in a **dog** following successful transplantation of DLA-identical fetal liver hematopoietic cells. There was no evidence of acute or chronic graft-versus-host disease. **Antibodies** to acetylcholinesterase receptor and immune complexes reactive with myoneural junctions were demonstrated, as well as focal inflammation with perifascicular and type 2 muscle atrophy. The **dog** responded to treatment with prednisolone and pyridostigmine.

L110 ANSWER 78 OF 84 EMBASE
on STN
ACCESSION NUMBER: 81089971 EMBASE
DOCUMENT NUMBER: 1981089971
TITLE: **Tumoricidal** responses in spontaneous **canine neoplasms** after **extracorporeal** perfusion over immobilized protein A.
AUTHOR: Terman D.S.
CORPORATE SOURCE: Dept. Med., Baylor Coll. Med., Houston, Tex. 77030, United States
SOURCE: Federation Proceedings, (1981) 40/1 (45-49).
CODEN: FEPRA7
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
016 Cancer
026 Immunology, Serology and Transplantation
LANGUAGE: English
AB I describe morphologic, histologic, immunohistochemical, and serologic changes in **dogs** with spontaneous breast adenocarcinoma, squamous cell **carcinoma**, hemangiopericytoma, and fibrosarcoma after extracorporeal perfusion of plasma over heat-killed and formalin-stabilized Staphylococcus aureus Cowans I (SAC), which was embedded in a membrane filtration system. In 12 **dogs** with breast adenocarcinoma, **tumor** necrosis was observed within 12 hours after perfusion; 24 hours after perfusion, multiple visible lesions in 6 of 6 **dogs** exhibited necrosis, but there was no reaction in uninvolved normal mammary tissue. In 8 **dogs**, healing of large ulcerated areas of cutaneous **tumor** was observed within 8 to 18 days after perfusion. Similar **tumoricidal** responses were observed in **dogs** with other **neoplasms** after SAC perfusion. **Tumor** cell necrosis observed within 4 hours after extracorporeal perfusion was associated with immunohistochemical deposits of IgG and C'3 and ultrastructural evidence of lytic lesions on **tumor** cell membranes. No **tumoricidal** effects were observed after perfusion over Staphylococcus aureus Woods (SAW) (nonprotein A bearing) in 3 **dogs** that previously or subsequently responded to SAC perfusion. No **tumoricidal** reactions were noted after phlebotomy of up to 50% of plasma volume in 6 **tumor** -bearing **dogs** that subsequently responded to SAC perfusion. SAC but not SAW perfusion was followed by increases in circulating **tumor** associated **antibodies** (TAA) for up to 48 hours after perfusion. Immune complexes increased after perfusion and remained elevated for 72 hours. Findings suggest that the acute **tumoricidal** responses are not due to mere removal of circulating immune reactants and may be initiated by TAA that are rendered operational after extracorporeal perfusion over SAC. The rapidity, specificity, and magnitude of the observed **tumoricidal** effects in various **canine**

neoplastic diseases suggests that this may have potentially broad-based therapeutic and biologic implications for **canine neoplasia**.

L110 ANSWER 79 OF 84 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 76004606 EMBASE
DOCUMENT NUMBER: 1976004606
TITLE: [**Immunochemical** approaches to immunotherapy]. PROCEEDINGS, XI INTERNATIONAL **CANCER** CONGRESS, FLORENCE 1974.
AUTHOR: Benjamini E.; Scibienski R.J.
CORPORATE SOURCE: Dept. Med. Microbiol., Sch. Med., Univ. California, Davis, Calif., United States
SOURCE: (1975) Vol. 1/- (327-332).
DOCUMENT TYPE: Book
FILE SEGMENT: 037 Drug Literature Index
016 Cancer
LANGUAGE: English
AB Many of the immunochemical approaches to **tumor** immunoprophylaxis and immunotherapy have proven to be both rewarding and promising. Although the mechanisms underlying the various reported phenomena are still not clear, several generalizations and cautious conclusions may be warranted. It is imperative that the individual be immunocompetent; individuals with greatly progressive **tumors** may not have a chance on immunotherapy due to generalized immune incompetence. Surgery or chemotherapy at the proper time in the course of the disease may not only be effective in decreasing the **tumor** load but may actually play an important role in restoring immune competence, thereby enhancing the probability for successful immunotherapy. It appears that certain chemical or enzymatic modifications of **tumor** cells or **tumor** cell products afford excellent vaccines for immunoprophylaxis and potential vaccines from immunotherapy. Although conclusive experiments are still lacking, it appears that chemical modifications of **tumor** cells or their products, with the aim of preferentially inducing cell mediated immunity without the concomitant induction of circulating **antibodies**, is of great potential for both immunoprophylaxis and immunotherapy. Such preparations merit further intensive investigation, especially when given in conjunction with nonspecific immunostimulants. It is worthwhile to reiterate a point made many times regarding immunotherapy, namely that it has potential in the management of **cancer** but mainly when used as an adjunct to other forms of therapy. It appears that while immunotherapy suffers from great limitations in controlling large **tumor** masses, its great potential lies in the control of possible residual **tumor** remaining after other forms of **cancer** therapy.

L110 ANSWER 80 OF 84 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 2001-374802 [39] WPIX
CROSS REFERENCE: 2000-072623 [06]
DOC. NO. CPI: C2001-114532
TITLE: Novel isolated **feline** interferon alpha proteins and nucleic acid molecules encoding them useful for regulating immune responses in **animals**, and for preventing and/or treating **autoimmune diseases** and allergic reactions.

DERWENT CLASS: B04 C06 D16
INVENTOR(S): WONDERLING, R S
PATENT ASSIGNEE(S): (HESK-N) HESKA CORP
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001040313	A2	20010607	(200139)*	EN	72

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2001019416 A 20010612 (200154)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001040313	A2	WO 2000-US32826	20001201
AU 2001019416	A	AU 2001-19416	20001201

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001019416	A	Based on	WO 2001040313

PRIORITY APPLN. INFO: **US 1999-451527 19991201**

AB WO 200140313 A UPAB: 20021216
NOVELTY - An isolated **feline** interferon (IFN) alpha protein (I) comprising a fully defined amino acid sequences of 189 (FeIFN alpha 189a), 189 FeIFN alpha 189b), 166 (FeIFN alpha 166a), 166 (FeIFN alpha 166b), 189 (FeIFN alpha 189c), 166 (FeIFN alpha 166c), 194 (FeIFN alpha 194d), 171 (FeIFN alpha 171d), 189 (FeIFN alpha 189e) or 166 (FeIFN alpha 166d) amino acids as given in the specification, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) preparation of (I);

(2) an isolated nucleic acid molecule (II), encoding (I) having a sequence of PS;

(3) a recombinant molecule comprising (II), operatively linked to a transcription control sequence;

(4) a recombinant virus comprising (II);

(5) a recombinant cell comprising (II);

(6) an isolated **antibody** (III) that selectively binds to (I);

(7) a therapeutic composition when administered to an animal regulates an immune response in the animal, comprising (I), a mimetope of (I), (II) or (IV) that selectively binds to (I); and

(8) identifying a compound capable of regulating an immune response in an animal, involves contacting (I) with a putative inhibitory compound under conditions in which, in the absence of the compound, the protein binds to IFN alpha receptor or inhibits proliferation of granulocyte macrophage-colony-stimulating factor (GM-CSF) stimulated TF-1 cell activity and determining if the putative inhibitory compound inhibits the

binding to IFN alpha receptor or inhibits the proliferation of GM-CSF stimulated TF-1 cell activity.

ACTIVITY - Immunosuppressive; antiallergic; **antitumor**; antimicrobial; antiinflammatory.

The antiviral activity of the five Chinese hamster ovary (CHO) cell-expressed **feline** interferon (IFN)- alpha subtype proteins was tested. Crandell **feline** kidney (CRFK) cells were treated for 24 hours with or without IFN- alpha tissue culture supernatants produced. The cells were then infected with the **feline** calicivirus and cytopathic effects induced by the virus were assessed 12 to 14 hours later. The cell layers were fixed in methanol, stained with crystal violet and examined under the microscope or processed for the MTT (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide) assay.

Each of the five IFN- alpha subtype proteins demonstrated anti-viral activity. Pre-treatment with any of the subtypes of IFN- alpha proteins resulted in significant reduction in the virus-induced cytopathic effect. Also, CHO cell-expressed and Escherichia coli-expressed a **feline** IFN- alpha subtype proteins inhibited granulocyte-macrophage colony stimulating factor-induced proliferation of TF-1 cells.

MECHANISM OF ACTION - Immune response regulator; gene therapy; IFN alpha protein function protein inhibitor.

USE - (I), (II), (IV), and an inhibitor of (I) as identified by the above method is useful for regulating an immune response in an animal (claimed).

(II) is useful for the recombinant production of (I), and for regulating an immune response in an animal when administered for gene therapy. (II) is useful for obtaining immunoregulatory nucleic acid molecules. (IV) is used as reagent in assays to detect an immunoregulatory protein (which is (I)), as reagents in assays to modulate cellular activity through (I) and/or as tools to screen expression libraries and/or to recover (I) from a mixture of proteins and other contaminants, and to target compounds to antigen present in cells.

Therapeutic compositions comprising (I), (II), (IV) or an inhibitor of (I) is useful for preventing and/or treating **autoimmune diseases**, allergic reactions, infectious diseases, **tumor** development, inflammatory diseases and/or graft rejection.

The isolated proteins, nucleic acid molecules and/or **antibodies** can also be used as diagnostic reagents. The compositions comprising the inhibitor or (I) are also useful for treating the above mentioned diseases.

Dwg.0/0

ABEX UPTX: 20010716

WIDER DISCLOSURE - The following are also disclosed:
(1) homologues of (I);
(2) allelic variant of (II);
(3) mimetopes of (I);
(4) fusion proteins comprising (I);
(5) multivalent target proteins comprising (I) attached to one or more compounds that can bind to a receptive molecule on the surface of the cell, where regulation of a immune response is desired;
(6) feline IFNalpha nucleic acid molecule including one or more regulatory regions, full length or partial coding regions; and
(7) nucleic acid molecules that are oligonucleotides hybridizing under stringent hybridization conditions with complementary regions of (II). The oligonucleotide molecules are useful as probes or primers, or as therapeutic reagents to inhibit feline IFNalpha protein production or activity, and to protect animals from disease.

SPECIFIC SEQUENCES - (II) has a fully defined sequence of 567 (S1)

(FeIFNalpha567a) (coding strand), 567 (S3) (FeIFNalpha567a) (complementary strand), 567 (S4) (FeIFNalpha567b) (coding strand), 567 (S6) (FeIFNalpha567b) (complementary strand), 498 (S7) (FeIFNalpha498a) (coding strand), 498 (S9) (FeIFNalpha498a) (complementary strand), 498 (S10) (FeIFNalpha498b) (coding strand), 498 (S12) (FeIFNalpha498b) (complementary strand), 567 (S13) (FeIFNalpha567c), 567 (S15) (FeIFNalpha567c) (complementary strand), 498 (S16) (FeIFNalpha498c), 498 (S18) (FeIFNalpha498c) (complementary strand), 582 (S19) (FeIFNalpha582d), 582 (S21) (FeIFNalpha582d) (complementary strand), 513 (S22) (FeIFNalpha513d), 513 (S24) (FeIFNalpha513d) (complementary strand), 567 (S25) (FeIFNalpha567e), 567 (S27) (FeIFNalpha567e) (complementary strand), 498 (S28) (FeIFNalpha498e), 498 (S30) (FeIFNalpha498e) (complementary strand) nucleotides as given in the specification (claimed).

ADMINISTRATION - Therapeutic compositions containing active compositions is administered as a controlled release formulation.
(I) is administered by subcutaneous, intradermal, intravenous, intranasal, intraocular, oral, transdermal and/or intramuscular routes. Dosages range from 1 mug-10 mg (preferably 10 mug-1 mg)/kg body weight. Genetic vaccine (naked nucleic acid) is administered by intramuscular, subcutaneous, intradermal, intranasal, oral and/or transdermal routes in dosages ranging from 1 ng-600 mug.
A recombinant virus vaccine comprising (II) is administered by subcutaneous, intranasal, intraocular, intramuscular and/or oral routes in dosages ranging from 1x104-1x108 plaque forming units per kg body weight of an animal. Recombinant cell vaccine that express (I) are administered in dosages ranging from 108-1012 cells/kg body weight.
EXAMPLE - Feline interferon (IFN)-alpha nucleic acid molecules were polymerase chain reaction (PCR) amplified from a feline cDNA library. Total RNA was isolated from a cat (kitten) mesenteric lymph node cells. cDNA was made from the RNA. The cDNA was used as a template to isolate a feline IFN-alpha nucleic acid molecule by PCR amplification.
Using 5'-ATGGCGCTGCCCTCTTCCTTCTTG-3' and that of the reverse primer was 5'TCATTTCTCGCTCCTTATCTTTTCTGC-3'. Five PCR products were generated and sequenced. These products were included, respectively, in Clones 1, 2, 3, 5 and 6. Clone 2 includes a feline IFN-alpha nucleic acid molecule that was represented as nFeIFNalpha567a, which has a fully defined sequence of 567 nucleotides (S1) as given in specification.
nFeIFNalpha567a encodes a protein containing 189 amino acids (S2), referred as PFeIFNalpha189a. The proposed mature protein denoted as PFeIFNalpha166a, contained about 166 amino acids, extending from residue 24 to residue 166 of (S2). The nucleic acid molecule encoding PFeIFNalpha166a was denoted as nFeIFNalpha498a which has a fully defined sequence of 498 nucleotides as given in the specification. Clone 3 includes a feline IFN-alpha nucleic acid molecule that was represented as nFeIFNalpha567b, which has a fully defined sequence of 567 nucleotides (S4) as given in specification. nFeIFNalpha567b encodes a protein containing 189 amino acids (S5), referred as PFeIFNalpha189b. The proposed mature protein denoted as PFeIFNalpha166b, contained about 166 amino acids, extending from residue 24 to residue 166 of (S5). The nucleic acid molecule encoding PFeIFNalpha166b was denoted as nFeIFNalpha498b which has a fully defined sequence of 498 nucleotides as given in the specification. Clone 1 includes a feline IFN-alpha nucleic acid molecule that was represented as nFeIFNalpha567c, which has a fully defined sequence of 567 nucleotides (S13) as given in specification. nFeIFNalpha567c encodes a protein containing 189 amino acids (S14), referred as PFeIFNalpha189c. The proposed mature protein denoted as PFeIFNalpha166c, contained about 166 amino acids, extending from residue 24 to residue 166 of (S14). The nucleic acid molecule encoding PFeIFNalpha166c was denoted as

nFeIFNalpha498c which has a fully defined sequence of 498 nucleotides as given in the specification. Clone 5 includes a feline IFN-alpha nucleic acid molecule that was represented as nFeIFNalpha567d, which has a fully defined sequence of 567 nucleotides (S19) as given in specification. nFeIFNalpha582d encodes a protein containing 194 amino acids (S20), referred as PFeIFNalpha194d. The proposed mature protein denoted as PFeIFNalpha171d, contained about 171 amino acids, extending from residue 24 to residue 166 of (S20). The nucleic acid molecule encoding PFeIFNalpha171d was denoted as nFeIFNalpha513d which has a fully defined sequence of 513 nucleotides as given in the specification. Clone 6 includes a feline IFN-alpha nucleic acid molecule that was represented as nFeIFNalpha567e, which has a fully defined sequence of 567 nucleotides (S25) as given in specification. nFeIFNalpha567e encodes a protein containing 189 amino acids (S26), referred as PFeIFNalpha189e. The proposed mature protein denoted as PFeIFNalpha166e, contained about 166 amino acids, extending from residue 24 to residue 166 of (S25). The nucleic acid molecule encoding PFeIFNalpha166e was denoted as nFeIFNalpha498e which has a fully defined sequence of 498 nucleotides as given in the specification.

L110 ANSWER 81 OF 84 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 2001-147178 [15] WPIX
DOC. NO. CPI: C2001-043518
TITLE: Recombinant **feline** interleukin-12, useful as immunostimulant and for treating e.g. viral infections in **cats**, and related nucleic acid constructs.
DERWENT CLASS: B04 C03 D16
INVENTOR(S): LEUTENEGGER, C; LUTZ, H; PEDERSEN, N; SCHROFF, M; WITTIG, B
PATENT ASSIGNEE(S): (MOLO-N) MOLOGEN FORSCH ENTWICKLUNGS & VERTRIEBS; (REGC) UNIV CALIFORNIA; (UYZU-N) UNIV ZURICH; (UYZU-N) UNIV ZUERICH; (LEUT-I) LEUTENEGGER C; (LUTZ-I) LUTZ H; (PEDE-I) PEDERSEN N; (SCHR-I) SCHROFF M; (WITT-I) WITTIG B
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001004155	A2	20010118	(200115)*	GE	35
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000066835	A	20010130	(200127)		
EP 1208115	A2	20020529	(200243)	GE	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
US 2003157059	A1	20030821	(200356)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001004155	A2	WO 2000-DE2263	20000708
AU 2000066835	A	AU 2000-66835	20000708
EP 1208115	A2	EP 2000-954321	20000708

		WO 2000-DE2263	20000708
US 2003157059	A1 CIP of	WO 2000-DE2263	20000708
		US 2002-41672	20020108

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000066835	A Based on	WO 2001004155
EP 1208115	A2 Based on	WO 2001004155

PRIORITY APPLN. INFO: **CH 1999-1259**
19990708

AB WO 200104155 A UPAB: 20010317
NOVELTY - **Feline** interleukin-12 (fIL-12) polypeptide (I) that is expressed, by recombinant gene expression in eukaryotic or prokaryotic cells, in the form of both polypeptide chains of subunits p35 and p40. These proteins are formed so that, in equimolar concentrations and in presence of an immunizing antigen, they can be administered to carnivores, particularly domestic **cats**.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for the nucleic acid construct (II) encoding fIL-12 containing sequences at least 95% identical with the p40- and p35-encoding sequences (A, 990 bp and B, 669 bp respectively), reproduced in the specification, as immunostimulant for immunization against infectious diseases and/or for treating infections or **tumors** in **felines**, especially domestic **cats**.

ACTIVITY - Immunostimulant; antiviral; **antitumor**.

Nucleic acid constructs that encode (II) are useful as adjuvants for prophylactic immunization against viral diseases and for treating diseases associated with a Th1 defect. **Cats** were immunized (using loaded gold particles, delivered from a gene gun) with:

(i) DNA encoding the gp140 antigen; or

(ii) as (i) but including DNA encoding fIL-12, at DNA dose about 2 micro g.

Three treatments were given at intervals of 3 weeks, and 4 weeks after the last treatment the animals were challenged with FIV at 25 times the 50% infectious dose. All **cats** of (i) became seropositive for **antibodies** against the transmembrane protein of FIV but only 1 of 4 **cats** in (ii), indicating complete protection in the three others (confirmed by absence of provirus from the plasma).

MECHANISM OF ACTION - IL-12 induces synthesis of interferon gamma (Th1 response).

USE - The **feline** interleukin-12 polypeptide is useful for carnivores, and particularly domestic **cats** in the following:

(i) as immunostimulant, for preventative or therapeutic use; and

(ii) treating **tumors** and **autoimmune diseases**, or diseases associated with a Th1 defect, especially pre-existing infections by **feline** immune deficiency virus (FIV), **feline** leukemia virus and corona virus.

Dwg.0/5

L110 ANSWER 82 OF 84 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 1999-493494 [41] WPIX
CROSS REFERENCE: 1983-707055 [28]; 1988-056484 [08]; 1989-130047 [17]; 1990-305017 [40]; 1990-348485 [46]; 1992-096889 [12]; 1992-175125 [21]; 1992-200174 [24]; 1992-268664 [32]; 1992-331718 [40]; 1992-349203 [42]; 1993-018128 [02]; 1993-026900 [03]; 1993-076502 [09]; 1993-243234 [30];

1994-263767 [32]; 1995-036113 [05]; 1995-366231 [47]; 1995-366385 [47]; 1996-187644 [19]; 1997-042857 [04]; 1997-043114 [04]; 1997-051904 [05]; 1998-321465 [28]; 1998-332054 [29]; 1998-332055 [29]; 1998-332145 [29]; 1999-610231 [52]; 2001-280989 [29]; 2002-040232 [05]; 2003-567445 [53]

DOC. NO. CPI: C1999-144491
TITLE: Recombinant **poxviruses** comprising exogenous DNA encoding antigenic determinants useful in **immunotherapy** to immunize against **cancers** and other diseases such as influenza, Newcastle Disease and rabies.
DERWENT CLASS: B04 D16
INVENTOR(S): PAOLETTI, E
PATENT ASSIGNEE(S): (HEAL-N) HEALTH RES INC
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 5942235	A	19990824	(199941)*		164

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
US 5942235	A	CIP of	US 1981-334456	19811224
		CIP of	US 1982-446824	19821208
		Div ex	US 1984-622135	19840619
		Cont of	US 1987-90209	19870827
		CIP of	US 1987-90711	19870828
		CIP of	US 1987-110335	19871020
		CIP of	US 1988-186054	19880425
		Cont of	US 1988-234390	19880823
		Div ex	US 1990-537882	19900614
		Cont of	US 1990-537890	19900614
		CIP of	US 1991-805567	19911216
		CIP of	US 1992-847977	19920303
		CIP of	US 1992-847951	19920306
		Cont of	US 1992-881995	19920504
		CIP of	US 1992-918278	19920722
		CIP of	US 1993-7115	19930120
		Div ex	US 1994-184009	19940119
		Div ex	US 1994-228926	19940414
		CIP of	US 1994-306259	19940913
			US 1995-458356	19950602

FILING DETAILS:

PATENT NO	KIND		PATENT NO
US 5942235	A	CIP of	US 4603112
		Div ex	US 4722848
		CIP of	US 4769330
		Div ex	US 5110587
		CIP of	US 5155020
		Cont of	US 5174993
		CIP of	US 5378457
		CIP of	US 5505941

CIP of US 5583028

PRIORITY APPLN. INFO: **US 1995-458356 19950602; US 1981-334456 19811224; US 1982-446824 19821208; US 1984-622135 19840619; US 1987-90209 19870827; US 1987-90711 19870828; US 1987-110335 19871020; US 1988-186054 19880425; US 1988-234390 19880823; US 1990-537882 19900614; US 1990-537890 19900614; US 1991-805567 19911216; US 1992-847977 19920303; US 1992-847951 19920306; US 1992-881995 19920504; US 1992-918278 19920722; US 1993-7115 19930120; US 1994-184009 19940119; US 1994-228926 19940414; US 1994-306259 19940913**

AB US 5942235 A UPAB: 20030820

NOVELTY - A recombinant poxvirus (X), comprising exogenous DNA encoding an antigenic determinant of a pathogen which is then expressed in vivo in infected host cells after administration to a patient and therefore induces an immunological response, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(i) a composition (Y) comprising (X) useful for inducing an immunological response in a host; and

(ii) a method (I) for inducing an immunological response in a host, comprising (X) (or (Y)) to a patient so that the antigenic determinant is expressed in vivo by the infected cells, therefore inducing an immunological response in the host.

ACTIVITY - Anticancer; Cytostatic; Viricidal

MECHANISM OF ACTION - (X) functions as a vaccine. The exogenous DNA encoding the antigenic determinant is expressed within infected host cells stimulating the patients immune system to destroy pathogens or **cancer** cells.

10 Beagle **dogs** (5 months old) and 10 **cats** (4 months old) were inoculated subcutaneously with either 6.7 (5 animals) or 7.7 (5 animals) log10 TCID50 (median tissue culture infective dose) of ALVAC-RG (an attenuated canarypox virus-based vector that was a plaque-cloned derivative of the canarypox vaccine, Kanapox, into which a rabies virus glycoprotein G gene had been inserted). 4 **Dogs** and 4 **cats** were not inoculated and acted as controls.

No adverse reactions were observed in either the **dogs** or the **cats** with either dose of vaccine. 4 of 5 **dogs** immunized with 6.7 log10 TCID50 had specific **antibody** titers 14

days post inoculation, and all **dogs** had titers 29 days post infection. All the **dogs** were protected from a rabies challenge which killed 3 out of the 4 controls. In **cats**, 3 of 5 animals receiving 6.7 log10 TCID50 had specific **antibody** titers on day 14 and all were positive by day 29 (although the mean titer was low at 2.9 IU (immunizing units)). 3 ofd the 5 **cats** survived a challenge which killed all controls. **Cats** immunized with 7.7 log10 TCID50 had **antibody** titers on day 14 and on day 29 the Geometric Mean Titer was 8.1 International units (no data given for the response to a rabies challenge).

USE - (X) may be used to vaccinate patients against a wide range of diseases and **disorders** depending on the type of antigen encoded by the exogenous DNA. (X) may be used to vaccinate against diseases such as rabies, influenza and Newcastle Disease. It is particularly useful for immunizing against **cancers**.

The poxvirus (X) also provides a means of manipulating lymphocytes and **tumor** cells for use in cell-based immunotherapeutic modalities for **cancer**.

ADVANTAGE - (X) provides an effective means for immunizing against a wide range of diseases. They also have enhanced safety compared to unattenuated viruses (attenuation reduces the virulence of the viruses) and known recombinant poxvirus vaccines. This increased level of safety reduces the possibility of a 'runaway' infection in the host and reduces the chance of transmission from vaccinated to unvaccinated individuals and contamination of the environment.

DESCRIPTION OF DRAWING(S) - The diagram shows a method for the construction of the plasmid pRW842 by the insertion of rabies glycoprotein G into the TK deletion locus of plasmid pSD513 (derived from plasmid pSD460 by the addition of a polylinker region) and generation of the recombinant vaccinia virus vP879.

Dwg.0/39

ABEX UPTX: 19991105

ADMINISTRATION - (X) may be administered by any suitable method (e.g. subcutaneous injection or intranasal spray) and may be administered with adjuvants to increase the immune response generated.

EXAMPLE - A gene encoding the rabies glycoprotein G under the control of the vaccinia H6 promoter (see Taylor et al., Vaccine 6, (1988)) was inserted into the TK deletion plasmid pSD513 (identical to plasmid pSD460 except for the presence of a polylinker region (see diagram). The polylinker region was inserted by cutting pSD460 with SmaI and ligating the plasmid vector with the annealed synthetic oligonucleotide VQ1A/VQ1B.

5' GGGAGATCTCTCGAGCTGCAGGGCGCCGGATCCTTTTTCT 3' (VQ1A)

3' CCCTCTAGAGAGCTCGACGTCCCGCGGCCTAGGAAAAAGA 5' (VQ1B)

This formed the vector plasmid pSD513. pSD513 was cut with SmaI and ligated with a SmaI ended 1.8 kilobase cassette containing the gene encoding the rabies glycoprotein G gene under the control of the vaccinia H6 promoter (see Taylor et al., Vaccine 6, (1988)). The resulting plasmid was designated pRW842. pRW842 was used as a donor plasmid for recombination with the NYVAC rescuing virus (vP866) which is an engineered vaccinia virus strain generated by the specific deletion of 18 open reading frames encoding gene products associated with virulence and host range. Recombinant vaccinia virus vP879 was identified by plaque hybridization using 32P-labeled DNA probes specific for rabies glycoprotein coding sequences.

L110 ANSWER 83 OF 84 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 1998-018049 [02] WPIX
DOC. NO. CPI: C1998-006643

TITLE: Enhancing immunological responses against **antigens** - using first recombinant vector containing gene encoding antigen and boosting with different vector encoding same antigen.

DERWENT CLASS: B04 D16

INVENTOR(S): CHAMBERLAIN, R S; IRVINE, K R; RESTIFO, N P; ROSENBERG, S A

PATENT ASSIGNEE(S): (USSH) US SEC DEPT HEALTH; (CHAM-I) CHAMBERLAIN R S; (IRVI-I) IRVINE K R; (REST-I) RESTIFO N P; (ROSE-I) ROSENBERG S A; (USSH) US DEPT HEALTH & HUMAN SERVICES

COUNTRY COUNT: 75

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9739771	A1	19971030	(199802)*	EN	41
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN					
AU 9726787	A	19971112	(199811)		
US 2001036928	A1	20011101	(200168)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9739771	A1		WO 1997-US6632	19970421
AU 9726787	A		AU 1997-26787	19970421
US 2001036928	A1	Provisional	US 1996-15893P	19960422
		Cont of	US 1999-171086	19990122
			US 2001-838987	20010420

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9726787	A	Based on	WO 9739771

PRIORITY APPLN. INFO: **US 1996-15893P 19960422; US 1999-171086 19990122;** US 2001-838987 20010420

AB WO 9739771 A UPAB: 19980112

Method for inducing an enhanced immunological response against at least 1 antigen in a mammal, comprises:

(a) inoculating the mammal with a first recombinant vector comprising a DNA vector and a gene encoding the antigen, and

(b) inoculating the mammal with a boosting immunisation with a second recombinant vector comprising a second DNA vector and the gene encoding the antigen.

Also claimed is a method of **immunotherapy** for treatment of a **cancer** patient, comprising:

(a) immunising the patient with a first recombinant vector comprising a first viral vector and a gene encoding a **tumour**-associated antigen (TAA), and

(b) boosting the patient with a second recombinant vector comprising a second viral vector and the gene encoding the TAA.

USE - The methods are used for generating an antigen-specific immune response capable of preventing and/or treating disease. They can be used for the **immunotherapy** against, e.g. **cancers**, **autoimmune disease** or infection by HIV, influenza virus, herpes simplex virus, human papilloma virus, equine encephalitis virus, hepatitis **feline** leukaemia virus, **canine** distemper, rabies virus, Chlamydia, Mycobacteria, Legionella, malaria, Babesia, Schistosoma, Toxiplasma, Toxocara **canis**, Aspergillus or invasive Candida.

ADVANTAGE - The use of 2 different recombinant vectors generates a strong cytotoxic T lymphocyte (CTL) and **antibody** response against the antigens.

Dwg.0/4

L110 ANSWER 84 OF 84 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN
ACCESSION NUMBER: 1987-050102 [07] WPIX
DOC. NO. CPI: C1987-021009
TITLE: Protecting **animal** against viral infection - by inserting gene coding for interferon-induced protein into **animal**.
DERWENT CLASS: B04 C03 D16
INVENTOR(S): HALLER, O; LINDENMANN, J; WEISSMANN, C
PATENT ASSIGNEE(S): (STAE-I) STAEHELI P
COUNTRY COUNT: 13
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 8700864	A	19870212	(198707)*	EN	72
RW: AT BE CH DE FR GB IT LU NL SE					
W: JP US					
EP 231374	A	19870812	(198732)	EN	
R: AT BE CH DE FR GB IT LI LU NL SE					
JP 63500800	W	19880324	(198818)		
EP 231374	A4	19891108	(199508)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 8700864	A	WO 1986-US1818	19860731
EP 231374	A	EP 1986-906512	19860731
JP 63500800	W	JP 1986-505748	19860731
EP 231374	A4	EP 1986-906512	

PRIORITY APPLN. INFO: **US 1985-761092 19850731**

AB WO 8700864 A UPAB: 19930922
Protecting an animal against viral infection comprises inserting into an animal a gene coding for an interferon - induced protein which is capable of protecting the animal against the viral infection.

Recombinant DNA molecule has a defined nucleic acid sequence and codes on expression for Mx protein and a polypeptide having a mol. weight of 80,000 Daltons that is immunoprecipitable with monoclonal **antibody** 2C12 and that is inducible in human peripheral blood lymphocytes, human fetal lung cells and human fibroblasts by interferon - alpha or interferon - beta, but not by interferon -gamma, and segments of the polypeptide.

USE - The method can be used for protecting an animal (including)

human) against influenza or other orthomyxoviral infection. Similarly animals may be provided with genese coding for interferon - induced proteins that have beneficial properties other than antiviral properties against orthomyxo virus, e.g. **anti-tumour** properties or anti-viral properties against viral infections such as picorna viral infections, such as foot-and-mouth disease virus, or paramyxovirus infections such as **canine** distemper or rinderpest affecting cattle. The polypeptide can be used for the prophylactic and/or therapeutic treatment of influenza and other viruses.
/19

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US 2000-563826 A2 20000503
US 2002-52323 A2 20020118
US 2002-116963 A2 20020405

ED Entered STN: 07 Mar 2003
AB The present invention relates to techniques of skin-targeted non-invasive gene delivery to elicit immune responses and uses thereof. The invention further relates to methods of non-invasive genetic immunization in an animal and/or methods of inducing a systemic immune or therapeutic response in an animal following topical application of vectors, products therefrom and uses for the methods and products therefrom. The methods can include contacting skin of the animal with a vector in an amount effective to induce the systemic immune or therapeutic response in the animal as well as such a method further including disposing the vector in and/or on the delivery device. The vector can be gram neg. bacteria, preferably Salmonella and most preferably Salmonella typhimurium. The topical vaccines can be used to treat and/or prevent infection and **cancer**.

L110 ANSWER 18 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:185083 HCAPLUS
DOCUMENT NUMBER: 136:226783
TITLE: Chelating agent and method of prevention and treatment of **cancer** and other diseases in animals
INVENTOR(S): Fernandez-Pol, Jose A.
PATENT ASSIGNEE(S): Novactyl, Inc., USA
SOURCE: PCT Int. Appl., 73 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 8
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002020486	A2	20020314	WO 2001-US27578	20010905
WO 2002020486	A3	20020704		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 6579891	B1	20030617	US 2000-657554	20000908 <--
AU 2001088788	A5	20020322	AU 2001-88788	20010905

PRIORITY APPLN. INFO.:

US 2000-657554	A	20000908
US 1995-581351	A2	19951229 <--
US 1996-26992P	P	19960920 <--
US 1996-24221P	P	19961022 <--
US 1997-843157	B2	19970411 <--
US 1998-127620	A2	19980801 <--
US 2000-182608P	P	20000215
WO 2001-US27578	W	20010905

OTHER SOURCE(S): MARPAT 136:226783
ED Entered STN: 15 Mar 2002
AB An antiproliferative, anti inflammatory, antiinfective, immunization agent of a metal ion chelating agent such as picolinic acid or derivs. thereof,

and methods of using the same. The agents chelate metals in metal containing protein complexes and enzymes required for growth, replication or inflammatory response. The prepns. can be administered systemically or for topical use. The prepns. have antineoplastic, antiviral, antiinflammatory, analgesic antiangiogenic and antiproliferative effects and are used in the treatment of warts, psoriasis, acne, skin **cancers**, sunburn, inflammatory responses, untoward angiogenesis and other diseases and in the prevention of sexually transmitted diseases such as genital warts, herpes and AIDS.

L110 ANSWER 19 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:946899 HCAPLUS
DOCUMENT NUMBER: 138:308
TITLE: Viruses for the treatment of cellular proliferative disorders
INVENTOR(S): Coffey, Matthew C.; Thompson, Bradley G.
PATENT ASSIGNEE(S): Oncolytics Biotech, Inc., Can.
SOURCE: U.S. Pat. Appl. Publ., 15 pp., Cont.-in-part of U.S. Ser. No. 708,663.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE	
US 2002187465	A1	20021212	US 2002-137194		20020501	<--
US 6596268	B1	20030722	US 2000-708663		20001109	<--
US 2002028195	A1	20020307	US 2001-965294		20010928	<--
US 6649157	B2	20031118				
US 2004057929	A1	20040325	US 2003-418290		20030418	<--
PRIORITY APPLN. INFO.:			US 1999-164878P	P	19991112	<--
			US 2000-708663	A2	20001109	

ED Entered STN: 13 Dec 2002
AB Methods for treating cell proliferative disorders by administering virus to proliferating cells having an activated Ras-pathway are disclosed. The virus is administered so that it ultimately directly contacts proliferating cells having an activated Ras-pathway. Proliferative disorders include but are not limited to neoplasms. Representative viruses include modified adenovirus, modified HSV, modified vaccinia virus, modified parapoxvirus orf virus, and modified influenza virus. Also disclosed are methods for treating cell proliferative disorders by further administering a immunosuppressive agent.

L110 ANSWER 20 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:674642 HCAPLUS
DOCUMENT NUMBER: 137:210939
TITLE: Methods of use of compounds which inhibit the stem cell factor signaling pathway
INVENTOR(S): Longley, B. Jack
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 22 pp., Cont.-in-part of U.S. Ser. No. 306,143.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002123031	A1	20020905	US 1999-474478	19991229 <--
US 6576812	B1	20030610	US 1999-306143	19990506
WO 2000067794	A1	20001116	WO 2000-US12405	20000505 <--

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-306143 A2 19990506 <--
US 1999-474478 A2 19991229 <--

ED Entered STN: 06 Sep 2002

AB The invention provides a method of preventing or treating in a subject contact dermatitis which comprises administering to the subject an amount of a compound capable of inhibiting the stem cell factor signaling pathway effective to prevent or treat contact dermatitis so as to thereby prevent or treat contact dermatitis in the subject. The invention also provides a methods of preventing or treating in a subject hyperpigmentation, asthma, cutaneous inflammation, anaphylaxis and bronchospasm, mastocytosis, **tumors** which express activated kit, and conception.

L110 ANSWER 21 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:595403 HCAPLUS
DOCUMENT NUMBER: 137:151141
TITLE: Novel human G protein-coupled receptors, their cDNA and protein sequences, other mammalian homolog related cDNA fragments and use thereof
INVENTOR(S): Bandman, Olga; Lal, Preeti G.; Tang, Y. Tom; Baughn, Mariah R.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 61 pp., Cont.-in-part of U.S. Ser. No. 156,513, abandoned.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002106655	A1	20020808	US 2001-895686	20010628 <--

PRIORITY APPLN. INFO.: US 1998-156513 B2 19980917 <--

ED Entered STN: 09 Aug 2002

AB The invention provides human G protein-coupled receptor (GPCR) proteins and their encoding cDNAs. They include two metabotropic glutamate receptors and some somatostatin and rhodopsin-like receptors and their related cDNAs and related fragments. It also provides for the use of the cDNAs, proteins, and antibodies in the diagnosis, prognosis, treatment and evaluation of therapies for neoplastic disorders and immune response. The invention further provides vectors and host cells for the production of the protein and transgenic model systems.

L110 ANSWER 22 OF 84 HCAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2002:276463 HCAPLUS
DOCUMENT NUMBER: 136:292947
TITLE: Prostate **cancer** marker PCAM-1 and **cancer** diagnosis by detection of gene expression or protein ligand binding and **cancer** therapy
INVENTOR(S): Stearns, Mark; Hu, Youji; Wang, Min
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 74 pp., Cont.-in-part of Appl. No. PCT/US2000/25981.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002042062	A1	20020411	US 2001-813380	20010321 <--
WO 2001021828	A1	20010329	WO 2000-US25981	20000921 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
WO 2002083081	A2	20021024	WO 2002-US8673	20020321
WO 2002083081	A3	20021212		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003100033	A1	20030529	US 2002-140602	20020507 <--
PRIORITY APPLN. INFO.:			US 1999-155865P	P 19990924 <--
			WO 2000-US25981	A2 20000921
			US 2001-813380	A 20010321
			US 2002-98992	A2 20020315
			WO 2002-US8673	A2 20020321

ED Entered STN: 12 Apr 2002
AB The invention relates to novel nucleic acids encoding a mammalian PCAM-1 gene, and proteins encoded thereby, whose expression is increased in certain diseases, disorders, or conditions, including, but not limited to, prostate **cancer**. The invention further relates to methods of detecting and treating prostate **cancer**, comprising modulating or detecting PCAM-1 expression and/or production and activity of PCAM-1 polypeptide. Further, the invention relates to novel assays for the identification of DNA-binding proteins and the double-stranded oligonucleotide sequences that specifically bind with them. The development.